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## THREE LITTLE KNOWN USEFUL AURAL AND NASAL OPERATIONS.[1]

By A. B. K. WATKINS, M.S. (London), F.R.C.S. (England), *Newcastle.*

THERE are various reasons why operations of proved worth are too little known.

Some have been used for a time, and then for various reasons fall into disrepute. When this happens, it is usually because there is some point in technique which has not been properly grasped, or because operators are unwise in the selection of patients to be treated. After that the proverb about giving a dog a bad name does the rest.

Some operations are little known because the surgeon performing them may not wish to publish them until he has collected a larger series.

It would not be difficult to draw attention to a number of valuable procedures which should be more widely known, but for this paper I will confine myself to the following three: (i) window resection for aural perichondritis; (ii) submucous resection of the inferior turbinates; (iii) transethmoidal pituitary approach.

[1] Read at a meeting of the Oto-Laryngological Society of Australia, Melbourne, August 16 and 17, 1954.

### Window Resection for Aural Perichondritis.

The diffuse type of aural perichondritis with effusion which supervenes after mastoidectomy with meatal flaps was a fairly uncommon complication of operation in past decades. Fortunately it has proved even less common in later years; but when it does occur the final result is disastrous, particularly in the male, whose hair is not long enough to hide the ugly deformity.

The first indications of this complication are pain and redness of the pinna. The pain may be severe and is usually continuous, lasting for days to weeks. The redness and slight swelling at first suggest erysipelas, but the onset is more gradual, the edge of the congested area is less sharp, and it never extends beyond the pinna. A high temperature or rigors are unusual.

The area involved extends slowly and the swollen skin over the area gradually becomes lifted on one or both aspects by an effusion.

Though cartilage is not dependent on blood supply, any such effusion when due to infection proves deleterious, and the cartilage undergoes necrosis and finally liquefaction. This usually progresses until organization takes place, and all that is left of the pinna is a small cauliflower-shaped mass of skin around the meatus (Figure I).

Surgery of the usual type consists of incision and drainage, with or without curettage or removal of portions of diseased cartilage, and seldom does such treatment defer the final deplorable result.

The reason for this is undoubtedly the fact that the cause of this variety of perichondritis is almost invariably the *Bacillus pyocyaneus*, and culture will prove the truth to those doubting Thomases who have seen this catastrophe develop in ears when the pus was neither green nor blue.

Figure I.
Healed aural perichondritis.

This organism is a robust bacterium which can grow and reproduce at room temperature, hence its menace as a nidus of wound infection in a ward.

As each new antibiotic has been presented to the public the surgeon has hoped that it might prove of paramount use in pyocyaneus infection. Though temporary claims have been made, so far all the hopes have been dashed to

Figure II.
Window resection for aural perichondritis. Note windows are curved and hidden as much as possible in folds.

the ground. However, chloramphenicol does have some deterring effect on the organism. New claims have just been made for some of the newer recruits, such as "Sulphamylon" and polymyxin, but I have not investigated results with them yet.

In 1929 R. C. Howard invented the operation of window resection for this complaint, but he also used it on aural hæmatomata. I cannot see the need for its use in the latter condition if infection has not supervened. In 1935 he reported five cases of acute post-mastoidectomy perichondritis, and claimed that all the patients did well after his operation, with little or no deformity. The operation consists in making a number of windows in the aural cartilages, the skin over the cartilage removed also being taken away, leaving the perichondritis of the posterior surface of the pinna exposed.

It might be surmised that a number of such windows extending beyond the area of effusion and perhaps nearly all over the pinna would result in extensive scarring and contraction. However, the actual contraction is surprisingly small and distorts the shape of the pinna so little that, unless it is compared closely with the normal one of the opposite side, it passes without notice.

The scarring can be hidden by making the windows as slots longer than they are wide, by arranging them in concentric rows and by placing them where they will be hidden by overhanging folds (for example, the helix or antihelix).

The method of making the windows is ingenious. A short incision through skin and cartilage is made, a septum dissector is passed in to undermine cartilage over the area for removal, and one blade of an opened

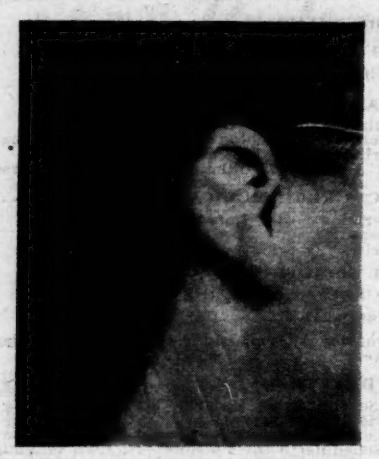

Figure III.
Healed aural perichondritis after window resection.

Grunwald's forceps is inserted, so that on closure the skin and cartilage are removed in one bite. To facilitate curving of the window, the forceps are not inserted as far as possible, but several short bites are made to make the bend possible (Figure II).

Subsequent treatment is, I think, relatively unimportant. I like to make a posterior splint for the pinna by soaking gauze in *Tinctura Benzoini Composita* and letting it set *in situ*, whilst I use *tulle gras* anteriorly to prevent the dressing adhering to the wound.

During the last fifteen years I have treated on these lines a larger series of patients than Howard, and in all cases the general shape of the pinna was more or less preserved—a very different state of affairs from that to which I had been accustomed (Figure III).

### Submucous Resection of the Inferior Turbinates.

We must all from time to time have been discouraged by the results of the ordinary surgical treatment of nasal obstruction due to hypertrophied inferior turbinates.

In one's attempt to steer betwen the Scylla of destroying too much turbinate with surgical removal, cautery, diathermy or chemical caustics, and the Charybdis of producing atrophic rhinitis by overdoing these treatments, one usually finds one has done insufficient. It is often not until one has repeated this treatment that one discovers one has overlooked the fact that sometimes the obstruction

is not due so much to mucosal deformity as to a deformity of the bone of the turbinate itself.

Fracture of the bony attachment seldom produces a permanent improvement, and gross removal involves grave risk of atrophic rhinitis.

It is in such a case that one of the forgotten operations is most useful. I cannot remember when or where I first heard of the operation, but I have used it for at least twenty-five years. The first reference to its existence, though without any further details, I find in the 1928 edition of Phillips's text-book, a volume which first came out in 1911.

Rhinologists have recently been reminded of this operation by Howard House (1951), though the bibliography he gives in his article does not go back before Harris (1936).

Once one conceives the idea of such an operation, not much knowledge is necessary to carry it into effect, provided that one does not fall into the trap of attempting it in a narrow nasal fossa without making more room by resecting the septum first.

The ideal case, and the one in which I have performed it most frequently, is that in which a hypertrophied inferior turbinate exists on the concave side of a deflected septum. Straightening the septum increases the obstruction on this side. In most cases the hypertrophy is mucosal and will adjust itself within three months, by which time, if compensation has not occurred, the cautery can be applied.

Sometimes, however, either at the septum resection or subsequently, the bony deformity is appreciated, and then submucous turbinate resection with preservation of the whole of the mucosa is indicated.

Under local anæsthesia, in which packs are placed on both aspects of the turbinate, an incision is made along the anterior border and the anterior part of the inferior turbinate.

The bone is here rough, strong and firmly adherent to mucosa. I manage with blunt dissection, even if it sometimes tears the mucosa. House recommends sharp dissection.

A St. Clair Thompson's speculum is inserted and used as in a septum operation. Progress is easier if the bone exposed is now removed with Blakesley's forceps (a half-scale size being most useful). The posterior part of the bone will be found to be rolled on itself like a scroll, but mucosa is less firmly attached and further dissection is easier.

The packing and after-treatment are carried out as after a septum resection.

I have found this operation so effective that I am driven to the conclusion that its fall into oblivion must be due to some operators attempting it without first assuring themselves of sufficient room. Under such conditions it can become such a difficult operation as to be most humiliating to its performer.

### Transethmoid Pituitary Approach.

We now come to an operation which is not well known because of lack of publicity. For this state of affairs perhaps I should crave your pardon, as I developed it and have used it since Ferris Smith gave me the key to the approach when he described his fronto-ethmoido-sphenoidectomy in 1934.

Previously the pituitary had been attacked by many routes. Hirsch used a submucous resection of the septum to go through the sphenoid to reach the pituitary. Cushing developed a slightly longer route along the septum, commencing as a sublabial incision. Fein used a transantral approach. The almost universally used modern route is the frontal one.

For some years I was interested in a shorter route through the ethmoid and sphenoid, but it was not until I had become acquainted with the finesse and blood-free technique of Ferris Smith's sinus operation that I was emboldened to extend his procedure and attack the pituitary.

In deciding on the route to use, one should first consider the routine frontal one. This is a moderately long route and whether one opens the dura right away and lets the brain fall away at once, in the hanging head position, or whether one defers opening it until one has dissected it to the lesser wing of the sphenoid, the operation is a major procedure and the intracranial cavity is opened, with the varying risks of trauma, infection and shock. The direction along which one approaches is such that when a tumour, as it frequently does, bulges into the sphenoid sinus, one can reach only the superior and posterior parts of the tumour.

I well remember assisting a world-famous cerebral surgeon in my younger days, when he demonstrated how he scooped out the whole of the tumour portion of the pituitary. The next day in the post-mortem room, a residual part of the tumour, which had been invisible from the frontal approach, was discovered. It was four or five times the size of the tissue he had removed.

This route therefore does not by any means give all the view which might be expected.

Further approach is restricted by the optic chiasma, underneath which one must proceed. Worse still, on either side are the stretched optic nerves. It is because of these that the surgeon selects the side with the worse vision on which the operate. He knows that he is quite likely to

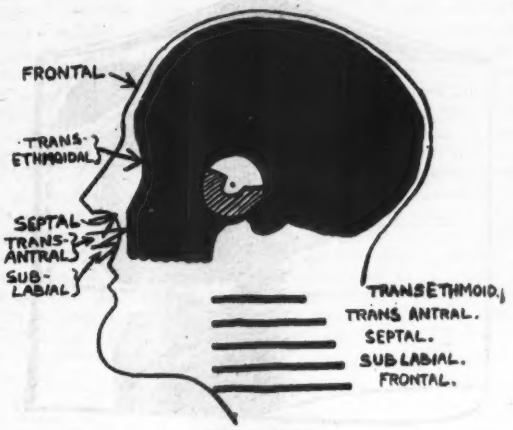


FIGURE IV.

Scale diagram to show relative lengths of approaches to pituitary fossa.

traumatize the nearer optic nerve. Not only this, he must be prepared deliberately to divide it to facilitate his advance—a tragedy, you will admit, for a patient with two defective eyes, whose main indication for operation is the preservation of his eyesight.

Fortunately, whatever the approach, not only is complete removal of the tumour not possible, but it is actually contraindicated, because life cannot be maintained without a certain amount of pituitary tissue. The various adenomata constitute the common tumours, and these infiltrate and fade away indefinitely into the rest of the pituitary. Therefore, in such cases, the operation resolves itself into removing enough tumour, often simply by suction, to decompress the chiasma and to allow as much time as possible before another decompression will become required. Fortunately these adenomata usually grow slowly, and a great many years of relief may be given before pressure symptoms recur. In the case of a frontal approach this involves even greater risks than before, because of intraarachnoid adhesions following the first operation.

When one compares my operation, one finds that there is such a short direct route that I have had spectators beside me who said that they could see everything without wearing a headlamp or mirror. The lengths of the various routes are shown in Figure IV.

My operation is surgically quite a minor procedure. Twice I have performed the operation under local anæsthesia only. Patients are usually well enough to get up

and walk about on the day after operation, and they usually say that their headache has been relieved and their vision improved immediately.

In cases in which there is no undue œdema of the eyelids, if it was not for the dressing, both fields could be plotted almost at once. Fields plotted on the available eye have sometimes shown improvement within three days. The intracranial cavity above the *diaphragma sellæ* is not opened, so technically the operation is only an extracranial one. Risk to the chiasma does not exist, whilst risk of injury to the optic nerves lateral to the sphenoid is present only when an inadequately trained surgeon is operating.

My operation allows any further growth of tumour to decompress itself by prolapsing into the naso-pharynx; should pressure symptoms again supervene in spite of this, the operation can be repeated even more simply as a direct transanasal manipulation.

The only point my operation concedes to the frontal route is that I confine mine to tumours of the pituitary fossa itself. It is not intended for and is quite unsuitable for the relatively rarer supradiaphragmatic tumours, such

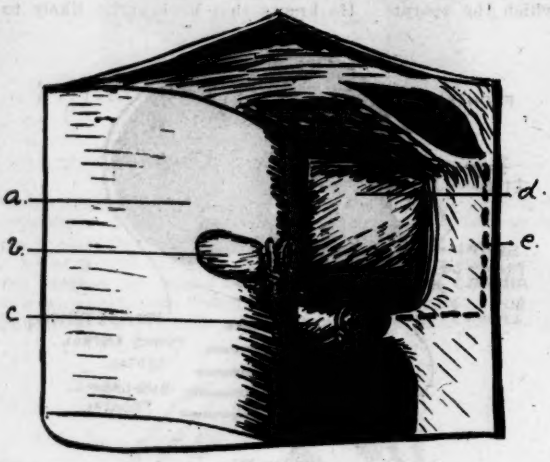


Figure V.

Completion of Ferris Smith's operation and commencement of Watkins's pituitary approach: (*a*) slotted guide of Ferris Smith retractor; (*b*) ligated stump of ethmoidal vessels engaged in slot; (*c*) sphenopalatine artery ligated in rolled divided mucosa; (*d*) right side of *sella turcica* seen through opened sphenoidal sinus; (*e*) line of Watkins's incision to remove bone and one mucosal layer of septum.

as cranio-pharyngiomata. It would not be likely to be of much benefit in the treatment of a malignant tumour, as evidenced by symptoms that suggest invasion of, rather than pressure upon, the chiasma tracts or optic nerves.

As most of my operation consists of Ferris Smith's sinus operation, I will commence by saying that I carry out his procedure *in toto*, except that there is no need to remove much of the floor of the frontal sinus, there is no need to strip out frontal sinus mucosa, and I am not in favour of his picric acid in acetone method of demarcating shreds of mucosa.

Having turned down the mucosa of the front of the nearest sphenoidal sinus after dividing the ligated sphenopalatine artery, I make a vertical incision in the septal mucosa about a third of an inch in front of the sphenoid face (Figure V). This is deepened by chiselling, and a submucous resection of the opposite side of the septum is made. The septal mucosa of the nearer side and bone is removed to the sphenoid face. The septal mucosa of the further side and the mucosa of the front of the opposite sphenoid are lifted forwards and displaced laterally together with the opposite spheno-palatine artery, which, being uninjured, does not need to be tied.

The whole of the face of both sphenoids is now exposed. It is broken with a sphenoid hook and widely opened up with punches. The powerful ones made for Ferris Smith are most useful (Figures VI and VII). The pituitary fossa will be seen either like a bas-relief or sometimes pushed forwards into close approximation to the anterior wall of the sphenoid sinuses. The floors and septum of the sphenoid sinuses are completely removed, as in Ferris Smith's operation.

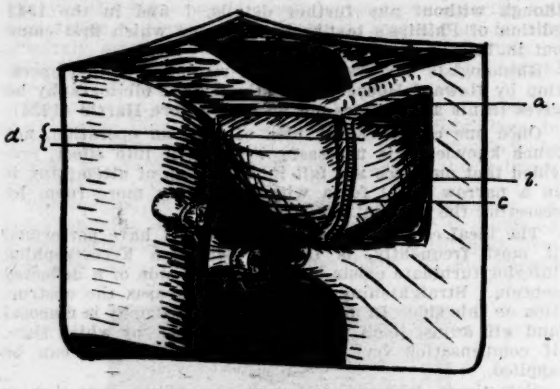


Figure VI.

Progress of transethmoid pituitary approach: (*a*) opening through septum (mucosa of left side and of anterior surface of left sphenoid face has been elevated and displaced to left); (*b*) opening into sphenoidal sinus; (*c*) unremoved portion of intersphenoid septum; (*d*) anterior surface of *sella turcica*.

The pituitary fossa now hangs into the posterior nasal fossa. It is usually of eggshell thickness, and can be easily cracked and removed with punches.

The tumour is now in full view within the firm pituitary capsule. This is opened by crucial incision. When this is done there is apt to be a certain amount of hæmorrhage for the first time, and often tumour tissue is extruded

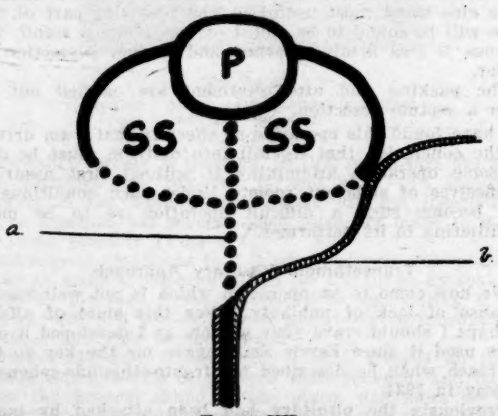


Figure VII.

Schematic view of operation from above: P, pituitary; S,S, sphenoid sinuses; (*a*) site of removal of bone and right-sided mucosa from septum; (*b*) displaced mucosa of septum and anterior surface of left sphenoid (containing unligated sphenopalatine artery).

(Figure VIII). According to the estimation one has made of the size of the tumour by X-ray examination, one removes more solid or semisolid tumour with the larger Blakesley's forceps, ceasing before one is uncertain whether one may have removed all the contents of the pituitary fossa.

The operation is concluded by applying a split thickness skin graft to the fronto-ethmoidal area on an inflated Ferris

Smith rubber bag, and the wound is closed with vertical sutures without drainage.

Sometimes a linear fibrous contraction makes the scar stand out slightly as a fold; but usually it heals as a flat surface, when it is difficult to see even in a patient who is not wearing spectacles.

The operation, I believe, fulfils the requirements of treatment of pituitary tumours; but I expect a hard fight to have it accepted, as I feel it should be, as the routine approach. I say this because patients with such tumours are usually referred to neurosurgeons. Such surgeons would have to learn more about practical procedure to be

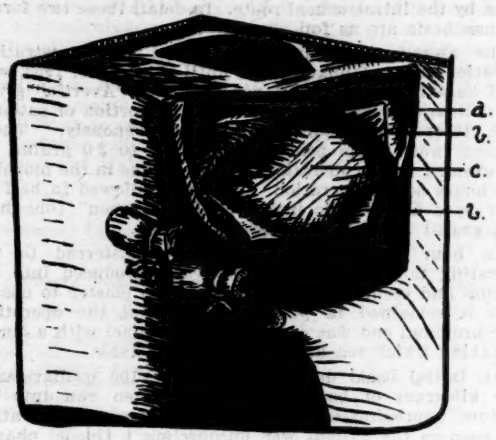


FIGURE VIII.

Completed transethmoid pituitary exposure: (*a*) opening in *sella turcica*; (*b*),(*b*) reflected flaps of parietal layer of dura lining sella; (*c*) bulging pituitary tumour.

able to use my method than would an oto-rhino-laryngologist, and the question would often resolve itself into the choice for the neurosurgeon of performing the operation by another route himself or handing it over to an oto-rhino-laryngologist to perform by mine.

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## SOME ASPECTS OF 500 FENESTRATION OPERATIONS.[1]

By G. C. HALLIDAY, H. D. RAFFAN AND R. G. MACKAY,
*Sydney.*

THIS report is based on 523 patients who had a fenestration operation performed at the Royal Prince Alfred Hospital, Sydney, by the three of us, who have worked as a team with the closest possible cooperation. The same operating theatre, anæsthetists and theatre assistants have been used throughout, while in the main an identical technique (Lempert) has been employed.

The first operation was performed on January 16, 1947, and the last in July, 1954.

[1] Read at a meeting of the Oto-Laryngological Society of Australia, Melbourne, August 16 and 17, 1954.

One of us (G.C.H.) gave a report on 85 cases at the Australasian Medical Congress (British Medical Association) in Perth in 1949, and a progress report of the first 50 of these has been included to indicate what the late prognosis may be. A further series of 50 consecutive operations completed on May 15, 1954, has also been listed, to serve as a comparison with the earlier series. These are set out in Appendix I. No attempt has been made to give a complete record of the entire series.

The larger the series of fenestration operations undertaken, the more apparent becomes the necessity for intensive and extensive practice on temporal bones or cadavers. Operations on temporal bones removed from a cadaver, though helpful, do not compare with operations on the complete cadaver.

Perhaps no other forms of cadaver surgery equips the surgeon so well as this procedure. Indeed, with the greater elasticity of living skin, it is actually easier to fashion the vital skin flap than in the cadaver, though of course the control of bleeding provides some initial difficulty—a difficulty which may obtrude itself even in experienced hands.

No doubt exists in our minds that there are basic requirements in a hospital if the best results possible are to be gained. Briefly these are as follows: (i) An operating theatre solely devoted to fenestrations. The preparation of an operating theatre for this work takes considerable time even in expert hands. Should two consecutive fenestrations be undertaken in one day, it is at least one hour before the theatre is ready for the second patient. Difficulties may arise, and an operation may take an hour longer than anticipated, so that the surgeon should have no worries that he is delaying the starting time of one of his colleagues. (ii) A permanent theatre sister or instrument technician, who should have sole control of all instruments and equipment, and assist in all operations. The importance of this member of the team cannot be over-stressed. Apart from her, only one other nurse is required in the theatre. (iii) An anæsthetist from a small team whose members cooperate closely with each other, and who is content to remain in the theatre throughout the operation.

### Selection of Cases.

Ideally, as in Boston, the selection of patients suitable for operation should be reviewed from many angles, the age factor, the type of employment, and the mental stability of the patient being taken into account. It is essential in all cases that the patient and his family should realize clearly that a percentage of failures is inevitable.

In our series the age has varied from eighteen to fifty-nine years.

### *History of Familial Deafness and Paracusis.*

In a dissection of the 100 cases which have been listed, hereditary deafness was present in 63 cases and absent in 30, and no information about it was available in seven cases. Paracusis was absent in 13 cases and not known in six cases. Of the cases in which paracusis was absent, there was no familial history in eight, but this appeared to have little effect in relation to the final results achieved, because a good result was obtained in 11 of these 13 cases. As paracusis is a subjective symptom there is a difficulty at times in establishing its presence or absence; but its absence may be disregarded if the clinical examination and discrimination tests otherwise give findings typical of otosclerosis.

### *Tuning Fork Tests.*

In our experience tuning fork tests are of extreme value. If they are carefully carried out, and if notations are made of increases and decreases in time duration of the Weber, Rinne and bone conduction tests, a result will be obtained which will bear an extremely close correlation to the patient's audiogram. In fact, good tuning fork tests followed by a discrimination test with masking of the other ear almost render an audiogram unnecessary.

Accurate results may be obtained only by the selection of matched tuning forks from C1 to C4, with a good broad base, and of such a construction that when the C2 fork is maximally struck it may be heard for approximately sixty seconds. In a recent visit to King's College Hospital, London, we found identical sets to those we have used for some years at the Royal Prince Alfred Hospital.

Dr. Silverman, of the Central Institute for the Deaf, St. Louis, a centre where very full audiometric investigations are made, told one of us that if one heard the voice of a deaf patient, took his history and then carried out a rough discrimination test, one could correctly assess his operability in 90% of cases.

*Audiometric Examination.*

At least two audiographic examinations have been made; the second one, prior to operation, should preferably be carried out with masking to the other ear, though this is not so important if the bone conduction thresholds are similar in each ear. On the other hand, if there is a 10 or 15 decibel difference in bone conduction of the essential frequencies, especially at 2048, it is most important that masking be used and that an equally careful discrimination test on the worse ear be made with masking of the better ear.

All are aware of the ideal audiogram which indicates operation in otosclerosis—no loss in bone conduction, and the loss in air conduction not greater than 50 decibels.

There are, in addition, two other patterns of audiograms seen in otosclerosis which indicate suitability for operation and merit discussion. The first shows a ski drop after 2048 or 3000, and the other is characterized by a moderate bone conduction loss, slightly more pronounced in the higher frequencies, and air conduction showing a parallel loss, with a difference of at least 25 decibels between air conduction and bone conduction in the essential frequencies.

Patients with the ski drop of the higher frequencies, which may occur at 2000 or 3000 cycles, appear to respond to operation almost as well as those patients who do not suffer from the loss in these high frequencies. In most of our successful cases the improvement in thresholds of the higher frequencies is never great, and in fact diminishes at times.

The second pattern presents a more difficult problem, as the prognosis at best is only second class; yet an improvement in this group appears even more gratifying to the patient than does the vastly greater benefit experienced by others.

It is generally conceded now that pure tone threshold audiograms are almost identical with those of speech audiograms in cases of conductive deafness, and it has not been our practice to take the latter type.

Discrimination tests can be practically carried out by use of the microphone attachment to the audiometer, or more simply by masking the other ear with a Bárány's noise box and dictating the P.B. list close to the patient's ear.

## Pre-Operative Care.

The ear for operation is examined and cleansed two days before operation. The patient is admitted to hospital on the day prior to operation and there has a complete physical examination, and X-ray films are taken of the mastoid regions. This routine has resulted in operations being deferred, and in several instances the operation has been abandoned permanently.

## Anæsthesia.

The first 400 patients were given "Avertin" with "Sodium Pentothal" in addition, as required. This permitted the free use of the diathermy current for coagulation; the deep narcosis following operation tended to prevent any post-operative increase in blood pressure, and generally speaking was satisfactory in most cases. Its disadvantages arose from a prolonged lowering of the blood pressure (necessitating the close attention of a special nurse for at least twenty-four to thirty-six hours after operation and causing a tendency to bed sores from pressure ischæmia), and more seriously in the plethoric adult, it tended to produce cyanosis and bleeding into the fenestra.

It is too early yet to say whether the prolonged immobilization from the "Avertin" anæsthesia was not superior to our present anæsthesia. None of our operations has been performed under local anæsthesia and sedation, as is the common practice in the United States of America.

The last 100 patients have been anæsthetized with pethidine (1%) and thiopentone sodium (5%) by intravenous injection, with the addition of nitrous oxide and oxygen given by the intratracheal route. In detail these two forms of anæsthesia are as follows.

The anæsthetic technique employed for fenestration operations from January, 1947, until September, 1953, was local anæsthesia and basal narcosis with "Avertin" given *per rectum*, supplemented in a small proportion of patients with thiopentone sodium given intravenously. These patients were given pentobarbitone, 1·5 to 3·0 grains, on the evening before operation, and 1·5 grains in the morning two hours before operation. This was followed in half an hour by a hypodermic injection of "Omnopon" (one-third of a grain) and scopolamine ($^1/_{100}$ grain).

An hour later the patient was transferred to the operating table, and a catheter was introduced into the rectum and securely fixed with adhesive plaster to ensure that it remained in position throughout the operation. The proximal end was connected to a funnel with a length of tubing which reached the foot of the table.

An initial basal dose of "Avertin" (100 milligrammes per kilogram of body weight) was then run into the rectum, approximately half an hour before the operation. As soon as the patient was unconscious a Guedel pharyngeal airway was introduced. Oxygen was delivered through another tube placed beside the airway.

Fractional amounts (one-quarter to one-third) of the basal dose were run into the rectum, through the catheter fixed in position, throughout the operation at intervals of half to one hour.

During the course of the operation, the average duration of which was two and three-quarter hours, one and three-quarters to two and a half basal doses of "Avertin" were generally required to maintain an adequate narcosis.

This technique called for meticulous attention to the airway, and in a proportion of the patients the lower jaw had to be held forward for long periods.

In the majority of cases this technique was entirely satisfactory, the blood pressure being maintained at 20 to 40 millimetres of mercury below normal. Thiopentone sodium was sometimes used as a supplement. It was administered at intervals in small doses by injection into the long saphenous vein over the medial malleolus. This was facilitated by the routine use in all cases of a continuous intravenous drip administration of glucose (4%) and one-fifth normal saline.

In a proportion of the later cases of this series "Vegolysin" was administered in doses of 25 to 100 milligrammes, to ensure that the blood pressure was lowered during the cutting of the window to minimize bleeding.

Since September, 1953, another technique has been adopted. The pre-operative sedation was the same as used previously. The technique now employed is as follows.

When the patient reaches the operating theatre the pharynx and larynx are sprayed with 5% "Xylocaine" solution. Anæsthesia is induced with 50 to 75 milligrammes of pethidine (1% solution) and 500 to 750 milligrammes of thiopentone sodium (5% solution) by intravenous injection. The larynx is intubated with a cuffed Magill tube liberally smeared with "Xylocaine" ointment (5%). Nitrous oxide and oxygen are then administered by a semi-closed technique. Additional pethidine (20 to 25 milligrammes) is given at intervals of three-quarters of an hour, as required.

It is advantageous to give the operating table a 15° to 20° footdown tilt to help minimize the bleeding.

### Fenestration Cavity Infection.

Infection of the fenestration cavity is best considered in two periods: (*a*) infection occurring within the first ten days after operation; (*b*) subsequent infection.

#### *Infection Within the First Ten Days.*

Though all our patients have been given as a routine penicillin, 100,000 units every eight hours, some disastrous infections have occurred. In 1951 several cases occurred in which the cavities became infected, the result being loss or partial loss of the drumhead, flap and hearing. Cultures were grown from material from the cavities, and in each case a *Staphylococcus aureus* resistant to penicillin was demonstrated.

After discussion with Dr. Edgar Thomson, director of the Fairfax Institute of Pathology, it was decided that after the first dressing all patients should be given "Aureomycin" by mouth in a dosage of one gramme per day in four divided doses. Moreover, it was arranged that at the first dressing cultures should be prepared from some deep portions, and Table I shows the results obtained in 69 cases.

TABLE I.

*Bacteriological Findings in 92 Cases from January, 1952, to July, 1954.*[1]

| Pathogens Isolated in Swabs from 64 Patients. | Number of Cases. |
|---|---|
| No growth, or non-pathogenic organisms .. .. .. | 45 |
| *Bacterium coli* or *Proteus vulgaris* .. .. .. .. | 6 |
| Group A hæmolytic streptococci[1] .. .. .. .. | 3 |
| Pathogenic *Staphylococcus aureus*[1] .. .. .. .. | 18 |
| *Clostridium welchii*[1] .. .. .. .. .. .. | 8 |
| *Bacillus subtilis* .. .. .. .. .. .. | 22 |

[1] Total number of infections with group A streptococci, pathogenic staphylococci and/or *Cl. welchii*, 24.

At the Staphylococcal Reference Laboratory, Fairfax Institute of Pathology, Royal Prince Alfred Hospital, under the direction of Dr. Phyllis Rountree, research bacteriologist, the phage types of the staphylococci isolated were determined and also the antibiotic sensitivities. These are set out in Table II.

From time to time nasal swabs were taken from members of the operating team. No staphylococci were found that could be directly implicated as the source of infection in these cases. It should be pointed out, however, that this does not exclude the possibility that other members of the operating theatre staff were in fact the source of the organism—and indeed this is almost certain. Steps are now being taken to investigate this point.

In the fatal case, well-established meningitis was present within three days of operation, and despite huge doses of penicillin (200,000,000 units of penicillin and "Aureomycin" in addition), the patient died seven days after operation. The organism grown on culture from the cerebro-spinal fluid was a *Staphylococcus aureus* found to be sensitive to penicillin.

Apart from the fatal case no other patient was ultimately affected deleteriously—a result achieved, we feel sure, by the use of "Aureomycin"; yet one cannot but be alarmed at the high incidence of infection, despite the utmost regard for asepsis and sterilization.

The other serious infecting organism was *Clostridium welchii*, all strains isolated being pathogenic in type. Patients infected with this organism showed varying degrees of toxæmia, some being extremely ill, while one or two showed but little effect. The infection did not in any case reach a stage at which gas was found in the surrounding tissues. In one case healing of the flap was delayed and the hearing possibly affected. The stay in hospital was not prolonged. These patients were treated with large doses of penicillin and, in some cases, with *Cl. welchii* monovalent antitoxin.

An interesting feature of these examinations was the failure to grow *Pseudomonas pyocyanea* on culture in any case.

It should be mentioned that the first dressing is carried out with the same scrupulous regard to asepsis as the operation itself.

#### *Infection After the First Ten Days.*

In our earlier cases, at a time when a more radical exenteration of mastoid cells was undertaken, many of our patients developed granulation tissue of varying profuseness; but since we have adopted the routine of insufflating the cavities with chloramphenicol or surgical "Aureomycin" powder and doing a minimum of dressings, granulations are now a rarity. It is preferable that all dressings should be done by the surgeon or the patient himself than that they should be entrusted to friends and relatives. As a number of patients are, or become, sensitive to chloramphenicol and "Aureomycin", any itchiness or increase in cavity secretion should be observed, and if it is present the powder should be forthwith abandoned.

The presence of sometimes persistent or recurrent discharge from the cavity has occurred in our hands as with others, and apart from the usual bacterial flora, including *Ps. pyocyanea*, there has been a not inconsiderable number of patients with mycotic infection.

We are grateful to Dr. G. C. Scantlebury, of Melbourne, for his suggestion that we might use ear drops of phenyl mercuric acetate (1/1000) in methyl alcohol, as this has been extremely successful. Though they cause some stinging pain in the acute phase of this infection, this minor objection rapidly disappears, and no apparent damage has been caused in any case.

TABLE II.

*Phage Types of Coagulase-Positive Staphylococcus Aureus Isolated from Fenestration Cavities.*

| Date. | Patient. | Ward. | Penicillin. | Streptomycin. | "Terramycin", "Aureomycin". | Chloramphenicol. | Phage Type. |
|---|---|---|---|---|---|---|---|
| 22/ 4/52 | A. | D2 | R.[1] | R. | S.[1] | S. | NT. |
| 25/ 6/52 | B. | GH4 | S. | S. | S. | S. | NT. |
| 1/10/52 | C. | GH4 | S. | S. | S. | S. | 42F. |
| 4/ 3/53 | D. | GH4. | R. | S. | S. | S. | 42F. |
| 1/ 4/52 | E. | GH4 | R. | S. | S. | S. | 29/42D/42H. |
| 22/ 4/53 | F. | GH5 | R. | S. | S. | S. | NT (*plus* group A streptococcus). |
| 29/ 4/53 | G. | GH4 | S. | S. | S. | S. | 29/42F. |
| 22/10/53 | H. | D2 | S. | S. | S. | S. | 47/47B/47D. |
| 30/10/53 | I. | GH5 | R. | R. | S. | S. | NT. |
| 6/11/53[2] | J. | D2 | S. | S. | S. | S. | 6/7/29/42D/47/52/52A (*plus* group A streptococcus). |
| 15/ 1/54 | K. | D2 | R. | S. | S. | S. | NT. |
| 10/ 3/54 | L. | D2 | S. | S. | S. | S. | 3A. |
| 8/ 4/54 | M. | D2 | S. | S. | S. | S. | NT. |
| 28/ 4/54[2] | N. | GH4 | R. | S. | S. | S. | 6/7/29/42D/47/52/52A. |
| 29/ 6/54 | O. | D2 | S. | S. | S. | S. | 6/7/47. |
| 2/ 7/54[2] | P. | GH4 | R. | S. | S. | S. | 6/7/29/42D/47/52/52A. |
| 22/ 7/54 | Q. | GH4 | R. | S. | S. | S. | 47/53/77. |

[1] R., antibiotic resistant; S., antibiotic sensitive (one culture not typed).
[2] Identical strains.

### Complications.

Until one week ago there had been no complications threatening life; but a patient developed a subdural abscess in the posterior fossa and died seven days after operation.

One patient has a minor facial paresis affecting her inferior labial muscle, while two other patients had transient facial pareses which rapidly and completely recovered within a few days.

There has been no case in which subsequent vertigo has seriously incommoded the patient, except in the immediate post-operative period.

A blue cystic swelling under the skin lining of the cavities has occurred in a number of cases, owing apparently to skin contraction taking place and causing a slow extravasation of blood. At times these swellings tend to spread to the region of the fenestra, and pressure on them causes vertigo. They should be needled or opened with extreme care.

Persistent and recurrent infections of the cavities have already been discussed *ad nauseam*, and they still have a large and irritating nuisance value.

Stenosis of the meatus has been avoided by an adequate removal of the fibrous tissue overlying the temporal muscle at the upper part of the wound. Though some cases in the early stages suggest that this may happen, as healing takes place an adequate meatus is left.

In no case has a subsequent operation for recurrent mastoiditis been necessary.

Extreme care must be taken to avoid a foreign body in the cavity, and the operation slip should always be read and inspected at the first dressing. Failure to do this in an early case or two soon showed it to be imperative.

### Skin Grafting.

Skin grafting has been undertaken in the last 100 cases, and even if the graft does not take in all cases it appears to leave the cavity cleaner and promotes earlier healing. In several cases during the last twelve months in which small perforations have occurred in the flap or periphery of the drumhead, we have overlaid them with fine skin grafts, and no resultant perforation has manifested itself.

We believe that there is a danger in the use of sea sponge packings to keep the skin grafts in their correct position. Any bleeding into the sponge makes an ideal culture medium, and we attribute our fatality to this cause.

### Causes of Failure.

In our opinion the greatest cause of failure is bleeding into the fenestra, resulting in fibrosis there. This is most likely to occur in plethoric subjects who are overweight and often appear short-necked, and above all when the foregoing characteristics are associated with hypertension.

Damage to the membranous labyrinth and flap no doubt plays its part; but there still remains a small group of patients whose hearing, despite a confirmed diagnosis of otosclerosis at operation and a completely satisfactory operation from a technical viewpoint, yet still fails to improve. Doubtless in some cases this is due to serous labyrinthitis, but certainly not in all.

Likewise, it is disappointing that increased facility in the performance of the operation does not appear to improve the percentage of good results.

### Final Assessment of Results.

As was mentioned earlier, we are appending the results of the first 50 and a final 50 cases. As will be seen, these results are very similar. Our criterion of a successful operation is that the air conduction loss of 2048 must not be greater than 30 decibels, and on clinical examination that the patient be hearing well by both conversation and whispered voice tests. Even though at the frequencies of 512 and/or 1024 the final post-operative level is below 30 decibels and the average decibel improvement is less than 20, nevertheless the patient's hearing is frequently excellent, especially if the threshold at 2896 is also at 30 decibels or less.

### Acknowledgements.

We are indebted to Dr. W. I. T. Hotten, Director of Anæsthesia at the Royal Prince Alfred Hospital, who has given and directed most of the anæsthetics. We wish to acknowledge with gratitude the great assistance that Dr. H. Selle, Superintendent of the Royal Prince Alfred Hospital, has afforded us since the inception of this work. Our thanks are due to the technical staff of the Fairfax Institute of Pathology for their exacting and prolonged task in our bacteriological investigations, and, above all, to Mr. Eric Bevan, the lay member of our operating theatre staff.

### Appendix.

*First 50 Fenestrations (G.C.H.), January 16, 1947, to February 5, 1948.*

There were 21 male subjects and 29 female subjects, of the following age groups: under twenty years, two; twenty to twenty-nine years, 10; thirty to thirty-nine years, 23; forty to forty-nine years, 10; fifty to fifty-nine years, two; age not known, three. The oldest patient was aged fifty-three years and the youngest nineteen years. The average age of the series was thirty-five years.

TABLE III.

| Decibel Gain. | Within Three Months. | At Four Years. |
|---|---|---|
| 0 to 9 .. .. .. | 2 | 4 |
| 10 to 19 .. .. .. | 11 | 19 |
| 20 to 29 .. .. .. | 24 | 13 |
| 30 and over .. .. | 13 | 1 |
| Not known .. .. | — | 13 |

A family history of deafness was present in 33 cases, absent in 14, and not known in three.

The decibel gain is shown in Table III.

Paracusis was present in 44 cases, absent in three and not known in three.

The results are shown in Table IV.

TABLE IV.

| Period. | Results. | | |
|---|---|---|---|
| | Good. | Poor. | Not Known. |
| Within 2 months .. | 47 | 3 | — |
| One year .. .. .. | 47 | 3 | — |
| Four years and over .. | 36 | 4 | 10 |

*Late 50 Fenestrations (G.C.H.), January 15, 1953, to May 13, 1954.*

There were 14 male subjects and 36 female subjects, of the following age groups: under twenty years, two; twenty to twenty-nine years, 15; thirty to thirty-nine years, 21; forty to forty-nine years, 8; fifty to fifty-nine years, three; not known, one. The oldest patient was aged fifty-one years and the youngest nineteen years. The average age of the series was 35·2 years.

TABLE V.

| Decibel Gain (Three Months). | Number of Subjects. |
|---|---|
| 0 to 9 .. .. | 2 |
| 10 to 19 .. .. | 12 |
| 20 to 29 .. .. | 29 |
| 30 and over .. .. | 5 |
| Not known .. .. | 2 |

A family history of deafness was present in 30 cases, absent in 16 and not known in four.

The decibel gain within three months is shown in Table V.

Paracusis was present in 37 cases, absent in 10 and not known in three.

The results within two months were good in 46 cases and poor in four.

*Ninety-Six Fenestrations (H.D.R.), April 2, 1950, to June 22, 1954.*

There were 72 female subjects and 24 male subjects, of the following age groups: under twenty years, two; twenty to twenty-nine years, 40; thirty to thirty-nine years, 24; forty to forty-nine years, 17; fifty to fifty-nine years, three. The oldest patient was aged fifty-five years and the youngest eighteen years, and the average age was thirty-one years. (No records are available for 10 patients.)

The decibel gain is as shown in Table VI.

TABLE VI.

| Decibel Gain. | Number of Subjects. |
|---|---|
| 0 to 9 .. .. | 7 |
| 10 to 19 .. .. | 19 |
| 20 to 30 .. .. | 48 |
| 30 and over .. .. | 12 |

Paracusis was present in 77 cases, absent in six and doubtful in three.

The results are shown in Table VII.

TABLE VII.

| Period. | Results. | |
|---|---|---|
| | Good. | Poor. |
| Within 2 months (86 cases) .. .. | 73 | 13 |
| After 12 months (76 cases) .. .. | 65 | 11 |
| After 2 years (54 cases) .. .. .. | 47 | 7 |

*First 33 Fenestrations (R.G.M.), November 15, 1951, to May 5, 1954.*

There were 12 male subjects and 21 female subjects, of the following age groups: under twenty years, one; twenty to twenty-nine years, 11; thirty to thirty-nine years, 15; forty to forty-nine years, six. The oldest patient was aged forty-eight years and the youngest nineteen years, and the average age of the series was thirty-three years.

TABLE VIII.

| Decibel Gain Within Three Months. | Number of Subjects. |
|---|---|
| 0 to 9 .. .. | 2 |
| 10 to 19 .. .. | 6 |
| 20 to 29 .. .. | 16 |
| 30 and over .. .. | 8 |
| Not known .. .. | 1 |

TABLE IX.

| Period. | Results. | | |
|---|---|---|---|
| | Good. | Poor. | Not Known. |
| Within 2 months .. | 29 | 3 | 1 |
| One year (17 cases) .. | 14 | 2 | 1 |

The decibel gain is shown in Table VIII.

Paracusis was present in 28 cases and absent in five.

The results are shown in Table IX.

## CLOSED ONE-STAGE FENESTRATION.[1]

By STEPHEN SUGGIT,
*Brisbane.*

HOLMGREN in 1948 advocated a closed fenestration operation and covered the fenestra with a previously prepared mucoperiosteal flap derived from the covering of the dome of the lateral canal.

In 1952 Garson described a post-aural approach without opening into the external meatus, the fenestra being covered with a piece of conjunctiva. The post-aural cavity was packed. A week later the packing was removed and the wound closed by secondary suture. Bauer, in 1953, went one step further. After applying the conjunctiva to the fenestra, he closed the post-aural wound without packing. A small drain was left for twenty-four hours.

I have followed Bauer's technique in the 23 cases described, except that I have covered the fenestra with a split-thickness skin graft from the skin immediately posterior to the incision, in place of conjunctiva.

In only one case I have succeeded in dissecting up a muco-periosteal flap as described by Holmgren, but I do not believe that the covering was adequate in extent. Garson makes the same comment.

The post-aural incision follows closely the crease behind the auricle and its upper part, and turns forward above the auricle. The mastoid cells are removed down to the aditus, and the facial ridge and the bridge are removed, care being taken to separate the soft tissues of the meatus down to the tympanic ring. On a couple of occasions I have buttonholed the meatal tube in removing a bone spicule, but no harm has ensued. I lower the facial ridge only sufficiently to get access. In all cases I have removed the incus. In some cases this may not be strictly necessary, but access is improved, and there seems little to be gained by retaining the incus. The fenestra is then made in the usual way into the lateral semicircular canal. I use a Shambaugh gold-polishing burr. The skin graft is then taken from the skin just posterior to the mastoid wound. This is done without moving the microscope or towels. I use a De Beers cataract knife with the pointed end removed, and take a split-thickness graft about one centimetre square. A little Ringer's solution is run into the cavity and the skin graft is floated off the knife, applied over the fenestra and moulded into the fenestral opening. Lempert has stressed the importance of invaginating the flap into the opening of the fenestra. It is easier to achieve this with a split-thickness graft than with the tympano-meatal flap of the classical operation. The thinner the graft, the easier it is to mould, but if it is too thin its viability is endangered.

Two pieces of "Calgitex" gauze about one inch long are packed over the graft, and suture of the post-aural incision is begun. When the incision is half closed the two pieces of "Calgitex" are removed and the graft is seen adhering to the bone surface. No packing is left in. The suturing of the incision is completed and a fine drainage tube is left in. The tube is removed in twenty-four hours. The only subsequent dressing is the removal of stitches on the seventh day, when the patient leaves hospital.

Fourteen of the 23 patients have been operated on six to ten months ago. Though it is too early and the numbers are too few to draw conclusions, these cases have been compared with my series of classical fenestrations at a similar period after operation. Table I shows the results of 93 classical operations, 82 performed by endaural route, and 11 by post-aural route in accordance with Simson Hall's technique. Patients of all ages and degrees of operability have been classed together.

At the end of six months not only is there a decrease of 17·2% in the number showing significant gain, but also many patients who had gains of 20 to 30 or 30 to 40 decibels are now in the lower category; these are respon-

[1] Read at a meeting of the Oto-Laryngological Society of Australia, Melbourne, August 16 and 17, 1954.

TABLE I.
*Results of 93 Classical Fenestrations.*

| Average for 500, 1000 and 2000 Double Vibrations. | 82 Lempert and 11 Simson Hall Operations. | | | | 59 Lempert Operations. | |
|---|---|---|---|---|---|---|
| | Two Months after Operation. | | Six Months after Operation or Longer. | | Two Years or Longer. | |
| More than 10 decibels worse | 2 | 2·2% | 3 | 3·2% | 3 | 5·1% |
| No significant loss or gain | 7 | 7·5% | 22 | 23·7% | 27 | 45·7% |
| 10 to 20 decibels gain | 18 | 19·4% | 25 | 26·9% | 10 | 17·0% |
| 20 to 30 decibels gain | 45 | 48·3% } 90·3% | 34 | 36·5% } 73·1% | 15 | 25·4% } 49·2% |
| 30 to 40 decibels gain | 18 | 19·4% | 7 | 7·5% | 3 | 5·1% |
| Over 40 decibels gain | 3 | 3·2% | 2 | 2·2% | 1 | 1·7% |
| Total | 93 | 100·0% | 93 | 100·0% | 59 | 100·0% |

sible for the number of significant gains falling to 49·2% at the end of two years. In others words, signs of closure of the fenestra are manifest as a rule by six months.

Table II shows the results of those cases in which the closed operation was performed. Though the figures are small, the results compare favourably with those of the classical operation. In three cases the fenestra has closed or was closing at four months after the operation; but of

TABLE II.
*Results of 23 Closed One-Stage Fenestrations.*

| Average for 500, 1000 and 2000 Double Vibrations. | Two Months after Operation. | Six to Ten Months after Operation. |
|---|---|---|
| More than 10 decibels worse | 0 | 0 |
| No significant loss or gain | 4 | 4 |
| 10 to 20 decibels gain | 9 } 19 | 4 |
| 20 to 30 decibels gain | 9 } 19 | 5 } 10 |
| 30 to 40 decibels gain | 1 | 1 |
| Total | 23 | 14 |

two other patients who at two months showed gain of less than 10 decibels, in one the gain increased to nearly 20 decibels and in the other to more than 20 decibels in the ensuing months.

Table III shows the results of both series of operations grouped as better or worse than the 30 decibels below the normal line. In this comparison the closed operation compares favourably with the classical fenestration.

TABLE III.
*Results Shown as Less or More than 30 Decibels Below Normal for the Average of 500, 1000 and 2000 Double Vibrations Six Months or More after Operation.*

| Hearing Loss. | Classical Operations. | Closed One-Stage Operations. |
|---|---|---|
| Loss of 30 or less than 30 decibels | 49 | 9 |
| Loss greater than 30 decibels | 44 | 5 |
| Total | 93 | 14 |

In six cases the classical operation has been performed on one ear and the closed operation at a later date on the other ear. Except in one case both operations were performed by myself. In one case the hearing came up well in both ears, and in both closure of the fenestra occurred at about the fourth month. In another case, of borderline operability, the classical operation produced a good result at one month. In the other ear at five weeks the hearing showed no significant gain; but when the patient was again examined at four and a half months the hearing had improved, though not yet to the level of the ear which had had the classical operation. In the third case the result of the closed operation is equal to the initial result of the previous classical operation on the other ear, and has been well maintained so far for three and a half months. This patient's ear previously treated by the classical operation showed signs of deterioration by three months.

The audiograms of the remaining three patients are shown in Figures I, II and III. The first of these (Figure I) is a medical student, who has an excellent result following a classical operation on the right ear performed by Dr. G. C. Halliday. With the connivance of Dr. Halliday and the patient, I performed a closed operation on the left ear. Vertigo, though not violent, was prolonged for several

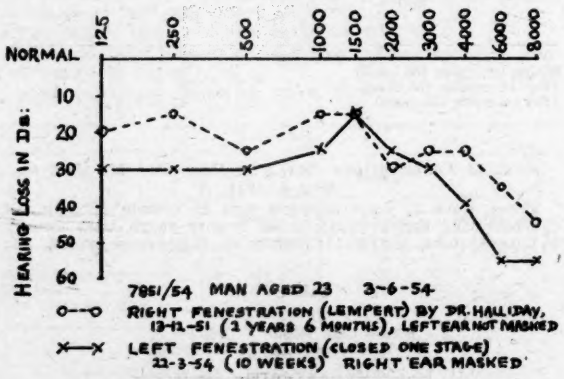


FIGURE I.

days; the patient assured me that it had been even worse after the previous operation. His hearing did not show signs of coming up for six weeks. The audiogram in this case was made ten weeks after the operation. The average pre-operation loss for 500, 1000 and 2000 d.v.s. was 55 decibels.

The second audiogram (Figure II) is the audiogram of the patient with a pre-operation average for 500, 1000 and 2000 d.v.s. of 60 decibels. He has a poor result in the left ear, presumably serous labyrinthitis, and the hearing has fallen slightly towards the pre-operative base line in the twenty months since operation. He has obtained a much better result in the right ear from the closed operation, as shown by his eight-months audiogram.

The third audiogram (Figure III) is that of a woman, who had a pre-operative average of 500, 1000 and 2000 d.v.s. of 45 decibels in both ears. She has a fair and useful result in the left ear, which was operated on by the classical endaural route two years and eight months ago. The result in the other ear at four months by the closed operation is a good deal better in the lower tones, but not

so good in the high tones; a more recent audiogram at eight months after the closed operation shows no significant change.

Post-operative vestibular disturbance has been on the average less than with the classical operations. Of the 23 subjects only two suffered from serous labyrinthitis; one is the medical student whose audiogram is shown in Figure I, and the other had a sudden attack of vertigo at four days. In both cases the hearing did not come up for nearly two months, but the end result was satisfactory.

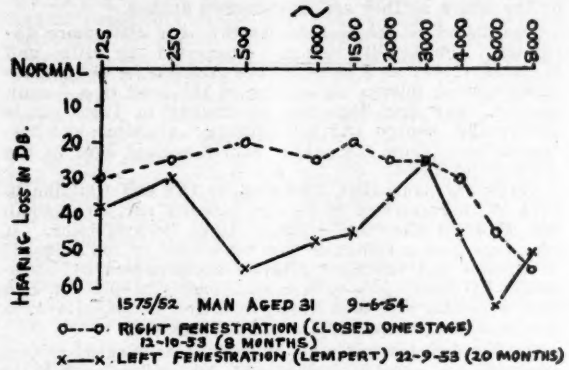


FIGURE II.

I have noted that delay in recovery of hearing is rather more frequent after the closed operation than after the classical operation. I think this may be due to the cavity being filled with granulations and blood clot rather than to serous labyrinthitis.

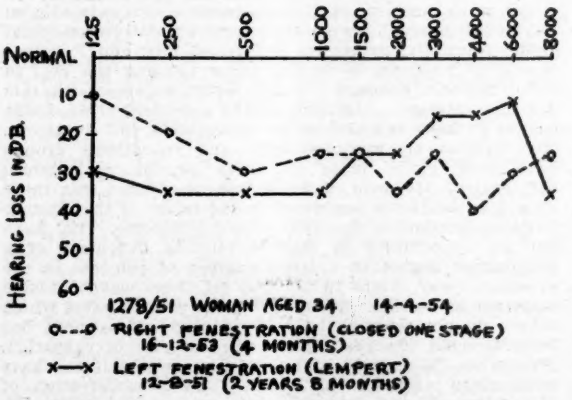


FIGURE III.

Cawthorne (1953) makes the following statement:

> The earlier operations by Jenkins, Holmgren and others were not successful because the wound of entry was closed up at the end of operation. This meant that air-borne sound waves which fell directly upon the ear drum were transmitted to the new fenestra and the round window. Consequently very little in the way of movement took place within the labyrinth.

The belief that it is necessary to separate the new fenestra from the tympanic cavity to obtain the hearing gain is widely held. The results of closed fenestrations I have described show that this belief is without foundation.

Though most of the cavities in the classical operation heal and are trouble-free, far too many continue to discharge for months, and even those that epithelize early may break down months or even years later.

*Pseudomonas pyocyanea* infection of the external meatus is endemic in the normal ear in Queensland, and the fenestration cavity with its thin vulnerable epithelium is an easy target.

In the closed cavity the mucoperiosteum of the middle ear probably relines the mastoid antrum as after a Schwartze operation in acute mastoiditis. Whether the skin graft over the lateral semicircular canal remains as such or is later replaced by mucoperiosteum I do not know; but I propose in the future revising the three ears in which the fenestra is closed.

As far as it goes, the closed operation shows promise, and it will be an advance if the patient's ear can be fenestrated without the infliction of a "radical cavity".

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## SOME ASPECTS OF THOSE CONDITIONS PROVISIONALLY DIAGNOSED AS MÉNIÈRE'S SYNDROME.[1]

By COLIN C. WARK,
*Brisbane.*

IT is now ninety-three years since Ménière first suggested the labyrinth as the site of the triad of symptoms which bears his name. However, there are descriptions in the literature which show that the condition was common throughout the years.

Thirteen years later our present idea of the function of the semicircular canals originated. Breuer (1875) first suggested that the utricle was stimulated by the position of the head in space. Bárány (1906) explained the significance of caloric stimulation and developed caloric and rotatory tests.

Since then much has been written, serving mainly to show the unsatisfactory state of knowledge of the morbid physiology and of the treatment of this condition.

In 1938 Hallpike and Cairns first demonstrated the pathological lesion in two patients who were known to have had the classical syndrome. We are, therefore, able to subdivide from the patients with vertigo presenting for investigation those whom we believe to have this lesion of the endolymphatic system.

But this is essentially an autopsy finding. Our knowledge of the normal physiology of the production of endolymph is speculative. Our knowledge of the abnormal physiology of Ménière's disease is even less certain. The condition itself does not cause death, and the labyrinth is not usually examined in the routine autopsy.

Clinically, the typical case with deafness, tinnitus and intermittent vertigo presents little difficulty in diagnosis. The hearing is often distorted just before an attack, and there may be a premonitory phase of increased tinnitus or deafness, while each attack leaves the hearing more impaired. However, the atypical case presents difficulty in determination of the site of the lesion. One important factor in this uncertainty lies in the definition of vertigo.

Brain has defined it as "the consciousness of disordered orientation in space".

The Oxford English Dictionary defines it as follows:

> Vertigo or giddiness is the disordered condition in which the person affected has a sense of whirling either of external objects or of himself, and tends to lose equilibrium.

[1] Read at a meeting of the Oto-Laryngological Society of Australia, Melbourne, August 16 and 17, 1954.

In the "Textbook of the Practice of Medicine", edited by F. W. Price, the following definition is given:

> "Vertigo", which by derivation means a "turning", is used to designate any movement or sense of movement or unsteadiness either in the individual himself (Subjective Vertigo), or in external objects (Objective Vertigo) that involves a defect, real or seeming, in the equilibrium of the body. It is a sensation of involuntary movement, either of subject or of external objects. It always involves a slight interference with consciousness, which in severe vertigo is often momentarily lost.

Most dictionaries define vertigo as giddiness, giddiness as dizziness, and dizziness as vertigo. Most otological textbooks avoid the definition altogether.

If we accept the narrower meaning as given by the Oxford English Dictionary—that is, with rotation and tendency to loss of equilibrium—then there are vestibular symptoms which are not truly vertigo—for example, the sensation of undulation persisting after sea travel. The "rotation" is interpreted by patients in a variety of ways.

If we take the broader definition, it includes many vague symptoms which are not labyrinthine—"blackout", and momentary loss of consciousness.

I suggest that we should take the narrower definition, realizing that vestibular conditions can produce symptoms other than the sensation of rotation, which are therefore not vertigo. The associated symptoms from overflow stimulation of the vagus nuclei are a fairly good indication that the condition involves the vestibular pathways.

The history is the most important factor in diagnosis, though it is often necessary to resort to direct questioning to get an admission of the sensation of rotation.

Pure tone audiometry shows perceptive deafness, which in itself is not indicative of Ménière's disease. The necessary information can be elicited much more quickly with tuning forks. The testing for recruitment by loudness balance or difference limen tests requires special equipment which is not always available. The presence of recruitment can usually be suggested by short-cut methods to indicate that the lesion is in the end organ.

It is my practice to carry out the caloric test by the method described by Cawthorne, Fitzgerald and Hallpike (1942). The main difficulty is to determine the end point even when the test is performed frequently. It is only too easy to fit the result to a provisional diagnosis unless there is an independent timekeeper. The other difficulty is in interpretation of results.

Taking the cases which are undoubtedly in the Ménière's disease group, we have many suggestions as to the ætiology: Knapp and Cheatle (1890), increased endolymphatic pressure; Mygind and Dederding (1932), abnormal fluid balance; Furstenberg and Lathrop (1934), sodium retention; Talbot and Brown (1935), potassium retention; Wright (1937), altered condition of endolymph due to inflammation; Crowe (1938), altered condition of endolymph due to physico-chemical factors; Hallpike and Cairns (1938), hydrops of endolymph. Then followed many suggestions to explain the hydrops, including allergy (Grove, 1941), vitamin *B* deficiency (Harris and Moore, 1940) and vasomotor conditions (Atkinson, 1946), and in 1952 Lempert *et alii* suggested herpetic vesiculations; but we are still uncertain.

The differential diagnosis is, firstly, that of the cases presenting Ménière's syndrome, as distinct from those that we group as presenting Ménière's disease. Pressure from blockage of the external auditory meatus by cerumen, or blockage of the Eustachian tube I find only too rarely; we can offer hope of rapid relief. Traumatic destruction of the labyrinth does not produce the remissions and relapses seen in Ménière's disease. Lesions proximal to the labyrinth usually do not cause both deafness and vertigo. Tumours of the eighth nerve rarely cause vertigo, since the progress is so slow and reorientation occurs without vertigo. Cerebellar lesions, vascular or neoplastic, produce labyrinthine symptoms without cochlear changes. Normal caloric reactions are the main safeguard against treating these as labyrinthine.

I find, among the cases referred as possible vertigo, that many do not from the history really suggest Ménière's syndrome; they are either cases of vertigo without deafness or frankly not cases of vertigo at all (disseminated sclerosis, the uncertainty or unsteadiness of *petit mal*, nausea and vomiting of migraine without deafness or vertigo, syncope or vasovagal attacks). The elderly patient often has tinnitus and deafness from presbycusis, and a cerebral vascular condition superadded perhaps causes difficulty in differentiation. But in all the hearing is not usually distorted, nor are the tinnitus and deafness affected by the attack as they are in Ménière's disease.

Two disorders are perhaps worthy of a little more discussion. Positional vertigo (suggested by Dix and Hallpike (1952) as a lesion of the utricle), in which transitory vertigo follows the placing of the head in a certain position, was first described by Bárány in 1921. It is paroxysmal, benign and self-limiting. Cochlear and vestibular tests show normal or nearly normal ears in the majority of cases.

Vestibular neuronitis, described by Dix and Hallpike in 1949, is characterized by vertigo, usually not paroxysmal, but with no abnormal cochlear signs or symptoms. It may vary from a feeling of "top heaviness" or "off balance" to sudden and transient seizures accompanied by blackouts. In these cases caloric abnormality is present, and it is evidently due in a high proportion to toxæmia from febrile illness or ear, nose or throat sepsis.

Recent experimental work on fluid balance suggests that treatment by restricting fluid and sodium intake, with or without ammonium chloride, makes only a transitory difference in the extracellular fluids. Readjustment is rapid, and suggests that this is illogical as continued therapy. Whether it has a place in the treatment of the acute attack, I propose to try to discover. My experience with this treatment gave some good results but a tendency to relapse. Theories based on potassium metabolism (Talbot and Brown, 1940) are open to this same objection, although recent work suggests that the potassium concentration in endolymph is much higher than in serum or cerebro-spinal fluid. Normally, potassium is intracellular, and I believe that it is unlikely to be the factor causing the rise in endolymphatic pressure. Some report success with this line of treatment. Atkinson (1949*a* and 1949*b*, 1950, 1953) has made many suggestions as to causation and treatment. His theories on vasoconstrictor and vasodilator groups have now given place to three groups of vitamin deficiencies. However, it would appear strange that there should be deficiency restricted to one factor of the vitamin *B* group producing the appropriate syndrome. We have had an opportunity to observe vitamin deficiency of a pronounced degree in a large number of subjects in the prisoner-of-war camps in Malaya, yet there was no undue incidence of vertigo. Atkinson reports great success which others do not seem to have reproduced. Treatment has been directed towards each of the theories of causation. Histamine, "Benadryl", and even these two combined have been given (Henderson, 1954). The administration of "Dramamine" and hyoscine is symptomatic treatment. The proprietary "Ronicol" given as a vasodilator is the latest; but I know nothing of its value, except a report of wide use in England.

What we are to do in the cases of post-traumatic or herpetic origin I do not yet know, other than to suggest sedation.

The intermittent nature of the condition would seem to suggest a neurovascular factor; but we have little proof, other than the results from sympathetic surgery and possibly from vasodilators.

The impression gained is that the success of any medical régime in the treatment of Ménière's disease depends on the enthusiasm and assurance with which it is carried out. We have a group of patients with a terrifying syndrome. To be stricken with acute vertigo, often with nausea and vomiting, usually with little warning, must give a feeling of uncertainty and insecurity even to the most stoical. It is my experience that any régime of medical treatment is likely to produce some improvement; but with the first relapse confidence is lost, and thereafter

the result is less certain. The more detailed and meticulous the treatment, the better. Perhaps this is the secret of Atkinson's success. Again, in my experience reassurance and explanation combined with mild sedation have in the long run produced as satisfactory a result as any other medical treatment.

The surgical treatment is directed either to destruction of the labyrinth in severe unilateral disease, with considerable success (Cawthorne, 1943), or to the sympathetic nervous system (Garnett-Passe and Seymour, 1948). Of the former I have had two cases. Of the latter I have had no experience except to see two patients in the early post-operative period. More recently, division of the *chorda tympani* (Rosen, 1953) does not seem to have any reasonable background, except perhaps in its secondary interruption of sympathetic fibres. Partial section of the eighth nerve is a more formidable procedure and preserves what is frequently distorted hearing (Dandy, 1941).

Injection (Mollison, 1931) or diathermy (Putnany, 1938; Day, 1943) has much the same effect as labyrinthectomy in destroying an ear in which hearing is hopelessly lost. There would seem to be an element of risk in these, and labyrinthectomy is more certain.

Whatever the ætiology, there is certainly a large functional element in most cases of Ménière's syndrome. There are even reports (Luscher, 1954; Zeckel, 1953) of patients successfully treated by psychotherapy.

The position is confused, and in both diagnosis and treatment we have not yet reached finality. No one of us sees enough of these patients to assess all suggested theories. Perhaps by pooling our experiences we may gain some ground.

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## SOME ASPECTS OF THOSE CONDITIONS PROVISIONALLY DIAGNOSED AS MÉNIÈRE'S SYNDROME.[1]

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THE provisional diagnosis of Ménière's syndrome carries with it the further responsibility of excluding other conditions presenting with some or all of the features commonly associated with this syndrome. The neurologist is frequently called upon to assist in this problem of establishing a diagnosis, the first essential of which is localization of the lesion. It is well recognized that the various components of Ménière's syndrome may result from lesions affecting widely separated areas within the central nervous system, and with this in mind I propose to discuss some aspects of differential diagnosis.

### Acoustic Nerve Tumours.

Acoustic nerve tumours, the most common of posterior fossa tumours, occur chiefly in the middle decades of life, at a time when the incidence of Ménière's syndrome is also high. Early diagnosis of this tumour depends on the clinician's awareness of it, the accurate evaluation of initial symptoms referable to the acoustic nerve and the trigeminal nerve, headache and ataxia, together with the finding of significant physical signs—deafness, impaired vestibular function, trigeminal sensory impairment and ataxia. It has been remarked (Edwards and Paterson, 1951) that these signs are so important that it is relatively unlikely that a patient presenting with deafness, who has not also impaired corneal reflex, cerebellar ataxia and nystagmus, will be suffering from an acoustic neuroma. At the same time recognition should be possible before all these signs are present, and a diagnosis made. Not infrequently the earliest symptoms are ignored by the patient and possibly by the doctor—symptoms such as mild deafness, tinnitus or vertigo. However, these symptoms are not always present in all cases, and Edwards and Paterson showed that when they were complained of, then they invariably appeared at an early stage, frequently some years previous to examination; in only 8% of cases were deafness and tinnitus not complained of. Vertigo, which was present in under 50% of their cases, was frequently only mild, sometimes brought on by sudden movement of the head. It is worth noting that there is a small group of patients whose complaint is of paroxysmal vertigo.

Most of these patients experience headache, either frontal or occipital, and like that resulting from other intracranial lesions it is frequently precipitated or aggravated by sudden exertion or change in posture. Headache may accompany vertigo of any origin, and it may play so important a part in the symptomatology of the Ménière complex as to arouse suspicion of eighth nerve tumour. The patient with Ménière's syndrome will complain of the feeling of a tight band round the head, possibly with suboccipital headache and neck pain; this is an unusua

[1] Read at a meeting of the Oto-Laryngological Society of Australia, Melbourne, August 16 and 17, 1954.

complaint in the patient with an acoustic tumour. It will be noted that all the symptoms so far mentioned—deafness, tinnitus and vertigo with headache—are prominent in Ménière's syndrome, so that any symptom indicating involvement of the trigeminal nerve is of importance. Although symptoms such as pain, numbness or tingling are uncommon, signs of involvement are frequent and of great importance. The corneal reflex will be found to be depressed or absent in the vast proportion of cases. In some cases the reflex is impaired bilaterally; but this should not necessarily be taken to indicate the presence of bilateral tumours, as the effect is produced by rotation of the brain stem. It is not unusual for the patient to present to the neurologist when ataxia is quite evident, and this may vary from slight clumsiness to gross instability. Non-coordination of a limb is much less common than is unsteadiness on the feet.

The application of simple tests to every patient complaining of the components of the Ménière syndrome would do much to aid in early diagnosis of these tumours and lessen the operative difficulties. Observe the gait for evidence of disequilibrium; the finger-nose test will reveal incoordination of a limb; the corneal reflex should always be elicited; and nystagmus will usually be found, and particularly as the condition progresses. Otologists themselves possess the very means for making a positive distinction between Ménière's syndrome and acoustic neuroma, for, as Hallpike has shown, loudness recruitment is present in Ménière's syndrome but not with acoustic nerve tumours. Further information may be obtained by careful radiographic examination of the skull, it being remembered that in skilled hands approximately 70% of eighth nerve tumours show changes.

### Lesions of the Cerebello-Pontine Angle.

One sees an occasional patient with meningo-vascular syphilis presenting with deafness, usually of rapid onset and painless without the accompaniment of other symptoms. Here examination of the cerebro-spinal fluid (which should be carried out in all cases in which there is no suspicion of raised intracranial pressure) will provide the diagnosis. Vascular abnormalities in this situation have been misdiagnosed as Ménière's syndrome. A basilar aneurysm has been seen presenting in such a fashion, only to be recognized as such with the occurrence of subarachnoid hæmorrhage and a subsequent vertebral arteriographic examination. Meningiomata of the lateral recess, and those which cause destruction of the petrous bone, including the glomus tumour, tend to involve the cranial nerves in less regular fashion than do eighth nerve tumours. However, the most important aspect of this discussion is to bring to mind the manifestations which indicate the presence of a lesion within the cerebello-pontine angle.

### Cerebellar Lesions.

It is because vertigo may be the initial complaint of a patient with Ménière's syndrome that I do not think it out of place to mention that true vertigo may result from a pure cerebellar lesion. Jefferson (1953) has shown that vertigo has been the presenting complaint of patients with tumours involving the floor of the fourth ventricle, the vermis and the cerebellar tonsil. Admittedly these patients do not complain of deafness; however, neither do some patients with Ménière's syndrome, and it is demonstrated only when they are examined. Furthermore, deafness may be a symptom of raised intracranial pressure, which itself is not uncommon in patients with the type of lesion mentioned above. Jefferson has adequately shown that we must no longer be reluctant to ascribe dizziness or vertigo to mid-line lesions of the cerebellum itself.

### Lesions of the Brain Stem.

In lesions of the brain stem it is obvious for anatomical reasons that deafness will be an infrequent symptom. However, temporary unilateral and sometimes bilateral deafness can result from brain stem lesions, more particularly vascular lesions. Everyone is familiar with the syndrome associated with thrombosis of the posterior inferior cerebellar artery in its fully developed form. In many cases the syndrome is incompletely developed, and here difficulties may arise. In the vast majority of cases vertigo is the prominent symptom. It is those features which accompany this vertigo which serve to localize the lesion to the brain stem; this is best seen in the above-mentioned syndrome, in which we find vertigo, deafness and tinnitus, unilateral ataxia of limbs, dysarthria, dysphagia and spino-thalamic disturbances—ipsilateral in the face and contralateral in the trunk and limbs. With involvement of vessels higher in the brain stem such manifestations as occular disturbances, nystagmus and tremor all occur. Several interesting points are worth mentioning in connexion with lesions in this situation; one point concerns bilateral deafness in case of basilar artery obstruction, usually a temporary phenomenon but on rare occasions permanent. Another point concerns the occurrence of vertigo in the elderly arteriosclerotic subject on flexing or extending the neck; here the mechanism is probably temporary basilar artery obstruction. A further point is to recall that vertigo may be a symptom of raised intracranial pressure; here it is usually paroxysmal and not infrequently associated with deafness. Disturbance of blood supply to the brain stem is the probable cause here.

### *Nystagmus.*

At this juncture I should like to mention a few points concerning nystagmus, confining my remarks to rhythmic nystagmus with its slow drift and quick return. It has become conventional to describe the direction of the nystagmus from the direction of the quick component.

If we presume that there is a slow drift to the right and a quick return to the left, then we speak of nystagmus to the left, and it will be more pronounced when the patient looks to the left side. If it is present only on looking to the left side, then this is first degree nystagmus to the left. Should the quick component be present on looking straight ahead, this is second degree nystagmus to the left, and if it occurs on looking to the right as well, then this is referred to as third degree nystagmus to the left. When testing for nystagmus we test first for spontaneous nystagmus, the patient looking ahead, to the sides and up and down. Then we test for positional nystagmus. The patient first sits up; he then lies back with the head turned to one side and the earlier routine search is carried out. Then the head is allowed to hang back and further observations are made. Next, sitting up once more, he looks straight ahead, and then the whole procedure is repeated with the head turned to the opposite side. It is important to repeat this routine if nystagmus is found, for positional nystagmus may be fatigable—that is, on repetition of the test no nystagmus is to be elicited. This observation has some considerable significance. Referring once more to spontaneous nystagmus, we have all witnessed this as a result of acute labyrinthine damage; for the first few days third degree nystagmus is present, then second degree nystagmus, and finally by ten to fourteen days slight first degree nystagmus remains and soon disappears. If spontaneous nystagmus persists for more than three weeks we should look elsewhere for the cause, as the end organ is unlikely to be at fault. A further obvious point which might be made is that nystagmus which is unaccompanied by giddiness will not be due to an end organ disturbance.

With regard to positional nystagmus, it is usually accepted that it results from a centrally placed lesion; but there is no doubt that positional nystagmus may result from a lesion both centrally and peripherally placed. In the case of centrally placed lesions the direction of the nystagmus may change with alteration in the position of the head, it will remain constant so long as that position is maintained, and furthermore it can be reproduced time and time again. When positional nystagmus is found but its direction does not change, then the lesion may be central or peripheral; but if fatigability can be demonstrated, then, as Cawthorne has shown, the lesion is not central. Peripheral positional nystagmus therefore occurs with the head in certain positions, usually to one side only, and it is fatigable. Cawthorne considers that the

lesion in these cases is in the static labyrinth, there being no associated cochlear involvement. Nystagmus of a vertical type has, in my experience, usually indicated a centrally placed lesion. The so-called "ataxic nystagmus"—that is, nystagmus in which the oscillations are more pronounced in the abducted eye—is the result of interference with the posterior longitudinal bundle and is therefore central in origin.

### Migraine.

The condition of migraine may seem to be fairly remote from this discussion; but there is no doubt that a small percentage of migrainous subjects are prone to brief episodes of vertigo as an aura to their attack. Usually it is accompanied by other more common manifestations of vasospasm elsewhere, and when it does occur it lasts for a shorter period than the more common visual or sensory aura. There is evidence that repeated vasospasm of vessels to the labyrinth may eventually give rise to permanent damage, and the later development of deafness and tinnitus is an indication of this.

### Epilepsy.

The question of epilepsy and vertigo I mention only to point out the difficulties that may arise in diagnosis, for it is well known that acute vertigo may produce sudden attacks of falling or unconsciousness. There are two points of value in distinguishing between acute vertigo and vertigo as an epileptic aura. Should vertigo be an aura, then it does not persist in the post-epileptic state, whereas the vertiginous sensation does persist should vertigo *per se* have caused unconsciousness. The other point is the characteristic post-ictal confusional state which should readily distinguish the two conditions.

### Vertigo Alone.

Finally there are those patients whose complaint is vertigo, and this may be unaccompanied by any other symptom of deafness or tinnitus, or by any sign suggesting a lesion of the brain stem or elsewhere. Not infrequently these patients are in the age group when degenerative lesions are uncommon and vascular lesions unlikely. Although the question of Ménière's syndrome may not seem to arise here, I believe one should include it within the differential diagnosis for the sake of completeness. I refer to that ill-understood condition vestibular neuritis, in which it is presumed that there is a selective lesion of the vestibular neuron or nucleus. Symptomatically the complaint is vertigo, paroxysmal, postural or chronic, and on examination one finds impaired caloric responses in the presence of preserved cochlear function, possibly positional nystagmus in the earlier stages and some evidence of disturbed equilibrium. The condition is commonly unilateral; it may not tend to recur; but it may be many months before a patient is fully recovered from the attack. One recognized cause of this condition is the administration of streptomycin. I have seen it occur in a severely diabetic subject with multiple peripheral nerve lesions. In this particular case it was bilateral, but not simultaneously so. There are doubtless many other causes of this condition which at present remain obscure; nevertheless it is fortunately a benign one, and only the course of time will provide the answer to the problem.

### Conclusion.

To make a provisional diagnosis of Ménière's syndrome, therefore, is only the starting point of a period of careful clinical evaluation, radiological investigation and otological examination, which should include audiometry, full caloric tests and, when indicated, assessment of the loudness recruitment, the necessity for excluding a treatable lesion being borne in mind throughout.

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## LIGATION FOR SEVERE EPISTAXIS.[1]

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ARTERIAL LIGATURE for severe epistaxis does not appear to be an operation which is performed with any degree of frequency; but if it is so performed at any of the major centres, the fact has not to date been recorded. The majority of text-books do not mention any operative procedures except that of local packing and transfusions; others dismiss the subject in their concluding sentences by mentioning ligation of the external carotid, and in desperate cases, of the internal carotid or the common carotid as well. A review of the literature revealed an amazing paucity of articles on the subject, and the fact that arterial ligature for severe epistaxis was a fairly rare procedure. From a paper by Arthur Spar and Olav Hallberg (1947) I was able to obtain most of the following references.

From a survey of world literature, McNight, of Texas, in 1926 found that the external carotid had been ligated for nasal hæmorrhage in 17 instances.

Abrahams in 1928 and Bricher in 1932 each reported a case of ligation of the external carotid for post-operative nasal hæmorrhage.

Hawthorn in 1930 and Johnson and Foster in 1933 each reported a case, the hæmorrhage being post-traumatic.

Barker in 1943 reported two cases.

Strauss in 1933 reported a ligation for idiopathic hæmorrhage in a pregnant woman.

Goodyear in 1937 ligated the anterior ethmoidal artery for nasal hæmorrhage, with its arrest.

Hirsch in 1936 described a ligation of the internal maxillary artery by a transantral approach in a case of nasal hæmorrhage. (Ligation by this method was first described by Seiffert, of Germany, in 1928.)

From 1930 to 1947 the external carotid artery was ligated for severe epistaxis in eleven cases at the Mayo Clinic. Of these cases, two were of post-operative origin, four of hypertensive origin, and the remainder spontaneous. During this period 106 patients had required admission to hospital.

In 1946, Weddell, MacBeth, Sharp and Calvert, of Oxford, reported seven cases in which control of epistaxis was obtained by ligation of the anterior ethmoidal artery.

There may possibly be other articles which have been overlooked, and which are of more recent origin; these have not been missed for want of searching.

The blood supply to the nasal cavity and sinuses is obtained from the corresponding common carotid artery which divides opposite the upper border of the thyroid cartilage into the internal and external carotid arteries.

The internal carotid artery in the cranium gives off the ophthalmic artery, which in the orbit provides the anterior and posterior ethmoidal arteries. These enter their respective foramina in the orbit and are ultimately distributed to the upper portion of the septum, the lateral wall of the nose, and the ethmoidal cells. Owing to the presence of the anterior communicating artery, a portion of the circle of Willis, the opposite carotid artery, or the vertebrals may also supply blood to these ethmoidal arteries. Therefore, a hæmorrhage from an ethmoidal artery may not necessarily be stopped by ligation of the corresponding internal or common carotid artery.

The remainder of the nasal cavity is supplied by the external carotid artery, the largest branch being the spheno-palatine artery, the terminal branch of the internal maxillary artery in the pterygo-palatine fossa. This enters the nasal cavity through the spheno-palatine foramen and supplies the lower portion of the septum and the lower lateral wall of the nose. It anastomoses in Little's area with the septal branch of the superior labial artery and the descending palatine artery, both of which derive their blood supply from the external carotid artery.

[1] Read at a meeting of the Oto-Laryngological Society of Australia, Melbourne, August 16 and 17, 1954.

The naso-pharynx is supplied by the pharyngeal and pterygoid branches of the internal maxillary artery, and these anastomose with branches of the external maxillary artery and the ascending pharyngeal artery.

It is stated that there is a fairly free anastomosis between the branches of the ethmoidal group on the one hand and the spheno-palatine branches on the other. In Case XV, after ligation of the external carotid artery bleeding still continued, but was so diminished that by forcible retraction of the middle turbinate the source of hæmorrhage could be seen—a ruptured and feebly pulsating spheno-palatine artery on the septum. This blood was probably coming from an ethmoidal anastomosis; but Hirsch maintains that it is from the opposite external carotid branches, and he therefore advocates ligation of the internal maxillary artery in the pterygo-palatine fossa by a transantral approach. Theoretically, even with ligation in that region, bleeding could still occur via the anastomotic septal branch of the superior labial artery.

It can therefore be seen that for control of severe epistaxis by operative procedures one has to determine where the bleeding is coming from, and then to decide which system to ligate. Interruption of the ethmoidal vessels will control bleeding which is coming from the superior regions of the nose, while interruption of the external carotid artery at the site of election should control an epistaxis from elsewhere, as would ligation of the internal maxillary artery. This latter procedure I have not employed, and I think it would be fairly difficult; but this place for ligation should be borne in mind if circumstances make ligation in the neck impossible or dangerous.

As I have shown, ligation of the internal carotid artery (while highly dangerous owing to cerebral complications) may not necessarily control hæmorrhage from ethmoidal vessels, and the same applies to ligation of the common carotid artery, which is not so dangerous.

Occasionally the source of the hæmorrhage is in the sinuses or the naso-pharynx, and interruption of the external carotid artery will control these sources if no other procedure does so.

A patient with such epistaxis consulted me in February, 1952. He was aged fifty-two years, and had been bleeding from the left nostril for about eight hours. On examination of the patient, blood was found dripping from the middle meatus, transillumination showed a dull antrum, and proof puncture produced a quantity of fresh blood. Bleeding luckily ceased on the instillation of one millilitre of adrenaline into the antrum, and X-ray films of the sinuses taken two weeks later showed no abnormality. The source of this hæmorrhage was probably in the antrum.

I think you will agree that the majority of epistaxes occur at Little's area, and although in some cases they may be very severe, they are readily controllable either by cautery or by packing. But it is when bleeding is occurring out of sight, and often out of reach owing to anatomical variations (septum and middle turbinates) that difficulty in control is experienced. The most common source of hæmorrhage in these cases is the spheno-palatine artery as it runs forwards and downwards on the septum towards Little's area. The bleeding point is as a rule hidden by the middle turbinate.

As far as possible, all patients with epistaxis are examined by the ear, nose and throat registrar on their arrival at the hospital. With a good light and a good sucker the nose is cleaned from clot and any packing, and the source of hæmorrhage, if possible, is determined. In most cases when the bleeding is severe it is extremely difficult to decide where it is coming from. Pressure over the ipsilateral common carotid often diminishes the hæmorrhage, and if complete occlusion can be obtained by pressure, epistaxis which does not diminish must be ethmoidal. If the source is visible, the point is then anæsthetized and cauterized with either galvanocautery or trichloracetic acid. If control is not obtained with this method, the region is packed and the patient is sent home.

Bleeding from high up in the nose or behind the front of the middle turbinate is dealt with by anæsthetizing the nose and packing it. Of the various things used for packs I prefer half-inch ribbon gauze impregnated with bismuth, iodoform and paraffin paste. One can leave this in the nose very much longer than anything else, as it keeps the nose clean and seems to stay in position better. "Vaseline" gauze is difficult to insert owing to its slipperiness, and furthermore I have seen it actually forced out of the nose by the pressure of blood building up behind it. I think it better also to pack from above downwards, leaving the floor of the nose last. In some cases in which control was not obtained, post-nasal packs were inserted; but I am not in favour of this, owing to the fact that blood can be forced past the packs, and to the danger that blood may enter the middle ear or the sinuses with subsequent infection. Unless the bleeding point is in the naso-pharynx the post-nasal pack acts only as a plug.

It appears that unless one can place the pack forcibly over the point of hæmorrhage, bleeding will continue if it is arterial and probably stop if it is venous or capillary.

Patients so treated are either sent home if the epistaxis is well controlled or kept in the casualty department if oozing is still in progress. The packs are removed in twenty-four to forty-eight hours, and further packs are inserted if necessary. If he is attending on a clinic day or operating day, the patient is examined either by myself or by someone in the clinic, and a further attempt is made to stop the epistaxis.

Owing to the shortage of beds of recent years these patients with recurrent or uncontrollable epistaxis are not admitted to hospital soon enough, and often patients are in and out of the casualty department for days at a time until they reach a stage at which their blood loss becomes serious and they are suffering from shock.

In the last three years 16 of these patients with so-called uncontrollable epistaxis were admitted to hospital under my care, and one was transferred to me, having been under the care of the physicians for some weeks—that is, 17 patients in all. Some of these patients had been under my care in the out-patient department, others had been attended by their own private doctors before their admission to hospital, and others had been treated in the casualty department.

On the patient's admission to hospital a blood transfusion is usually given to correct the blood loss, and the nose is repacked if the patient has not been in our hands before his admission. The pack is removed in twenty-four to forty-eight hours if bleeding has stopped, and the nose is repacked if hæmorrhage recurs. When bleeding is still going on although packs are *in situ*, as a rule repacking is carried out in twenty-four hours, or sooner if necessary.

Of the 17 cases, in 10 hæmorrhage had stopped or was only slight for a time on removal of the packs after the blood transfusion, and the patients were discharged from hospital; but two had to be readmitted after about a week, in each case with a fresh hæmorrhage. Fourteen of these 17 patients were aged over forty-five years, nine being males. Vessels were palpable in 13, 15 were hypertensive, one suffered from familial multiple telangiectasia, and two appeared to be otherwise perfectly healthy.

Nine patients were operated on, the external carotid arteries being ligated in all; the anterior ethmoids in addition were ligated in three cases, in one of which the operation was elective.

Compared with other reports and figures, this appears to be an unduly high proportion of ligations; but the first three operations were necessary for desperately ill patients, in whom control by other means could not be obtained. In some of the latter cases the hæmorrhage might have stopped had we devoted the time and blood, as we had to with the earlier cases; but even in some of these cases packing was carried out repeatedly up to fourteen days and more.

### Reports of Cases.

The following cases are in chronological order.

Case I.—The patient was a woman, aged fifty-six years, who for some weeks had been under the care of one of the physicians. She was suffering from gross familial multiple telangiectasia of the cavernous type, present on the face

and on the mucosa of the septum and turbinates. She had been in hospital for some weeks, and repeated attempts had been made with packing and cautery to stop the epistaxes, which were bilateral. During that period, she had received 20 pints of blood, which brought her total in four years to seventy-two transfusions. She had had several courses of deep X-ray therapy without effect. I ligated the external carotid artery and anterior ethmoid on one side, and a week later ligated the vessels on the opposite side. Since then (three years ago) she has had some small epistaxes, which have not been sufficient to bring her to hospital.

CASE IV.—The patient was a woman, aged seventy-one years, who came from the country and had been having recurrent epistaxes for three months despite frequent pack-

TABLE I.

| Case Number. | Age. (Years.) | Sex. | History. | Blood Pressure. (Millimetres of Mercury.) | Vessels. | Hæmoglobin Value. | Blood Given. | Treatment. |
|---|---|---|---|---|---|---|---|---|
| I | 56 | F. | Familial telangiectasia with repeated epistaxes over last four years. Had 52 transfusions in that period. | 142/82 | Cavernous telangiectasia of face and nose. | — | 20 pints. | Repeated packing and cauterizations. Deep X-ray therapy. Bilateral ligation of external carotids and anterior ethmoids. |
| II | 64 | F. | Epistaxes for last 5 days on and off despite repeated packing. | 180/110 | Thickened. | 67% | 1 pint. | Nose repacked, patient given transfusion. Bled slightly on removal of packs. Topical thrombin spray. |
| III | 60 | M. | Recurrent epistaxes for 6 days. | 150/96 | — | 66% | — | Pack removed in two days. No further bleeding. |
| IV | 71 | F. | Recurring epistaxes for 3 weeks despite packing repeatedly. | 140/80 | Thickened. | 81% | 2 pints. | Nose repacked in operating theatre, transfusion given. Recurrent hæmorrhages despite repacking. |
| | | | | 200/100 | — | 40% | 2 pints. | On sixth day sudden severe hæmorrhage, patient became comatose; transfusion. Ligation of external carotid artery and anterior ethmoid artery. |
| V | 74 | M. | Recurrent epistaxes, severe over last 24 hours. | 170/100 | Not thickened. | 30% | 3 pints. | No hæmorrhage after pack inserted in casualty department. Two small hæmorrhages on removal in forty-eight hours. Subsequently developed "a chest". |
| VI | 78 | M. | Severe epistaxis for 24 hours. | 190/105 | Thickened. | 73% | 1 pint. | Nose packed in ear, nose and throat theatre. Small amount of oozing when packs removed in forty-eight hours. |
| VII | 69 | M. | Severe epistaxis over 16 hours. | 90/60 | Thickened. | 72% | 2 pints. | Nose packed, and no further bleeding on removal in forty-eight hours. |
| VIII | 49 | F. | Severe epistaxes for last 4 days. | 170/85 | Slightly thickened. | 74% | 2 pints. | Nose packed, bleeding controlled. Bleeding occurred on removal of packs and would not stop. |
| | | | | | | | 2 pints. | After six days patient was given more blood and external carotid artery tied. |
| IX | 65 | F. | Recurrent epistaxes for last 9 days. Under treatment for hypertension. | 130/95 | Thickened. | 65% | 2 pints. | Epistaxis stopped with packs, but recurred on their removal. Nose repacked and bleeding stopped sufficiently to allow application of galvanocautery to point high up in nose. |
| X | 52 | F. | Epistaxes for 3 days, not controlled by packing. | 100/90 | Thick. | 80% | 1 pint. | Could not be controlled with packing. External carotid tied. No further trouble. |
| XI | 56 | M. | Recurrent epistaxes over a year; hæmorrhage occurring for 10 to 12 days. | 190/130 | Thick. | 64% | 1 pint. | Could not control hæmorrhage with packing. External carotid ligated. No further trouble. |
| XII | 28 | F. | Rheumatic fever 3 years previously; bled from spots on skin. | 100/60 | Normal. | 46% | 3½ pints. | Nose packed and bleeding controlled. No further bleeding on removal of pack. |
| XIII | 45 | F. | Recurrent epistaxes over 20 years. | 180/90 | Thick. | 65% | — | Cautery with trichloracetic acid, without success. Nose packed and no further bleeding on removal. |
| XIV | 40 | M. | Epistaxis and early quinsy; readmitted to hospital with severe epistaxis. | 150/100 | Thick. | 74% | — | Stopped during treatment for quinsy. Could not control hæmorrhage with packing. External carotid artery ligated. |
| XV | 65 | M. | Severe epistaxis for some days. | 120/70 | Thick ++. | 70% | 3 pints. | Given transfusion and nose repacked on a number of occasions. Finally hæmorrhage controlled and patient discharged after seven days' treatment. Could not control hæmorrhage with packing. External carotid artery ligated, and small pack applied over bleeding point. |
| | | | Readmitted 6 days later with recurrence. | 148/80 | — | 38% | 4 pints. | |
| XVI | 29 | F. | Severe epistaxis for 3 days. | 140/110 | Normal. | 60% | 2 pints. | Could not control epistaxis with packing and transfusions. External carotid ligated. |
| XVII | 49 | M. | Severe epistaxis for 3 days. | 148/82 | — | 86% | 3 pints. | The nose was repeatedly packed, but epistaxis recurred. Patient had a sudden severe epistaxis, and the external carotid artery was ligated. Transfusion of one pint after operation. |
| | | | | | | | 1 pint. | |

ing. She was admitted to hospital and her nose was packed. Over six days, during which time intermittent hæmorrhage continued, two pints of blood were given. The hæmorrhage was from high up in the nose. On the sixth day she had a very severe hæmorrhage, and when examined by me was unconscious and appeared to be dying. The transfusion and resuscitation unit gave her two pints of blood, corrected her electrolytic balance and brought her out of her coma, when hæmorrhage started again. She was taken to the operating theatre and had the external carotid tied, and as there was still some oozing from the upper part of the nose, the anterior ethmoid was then ligated. The nose was dry when she left the theatre, and she has had no recurrence since.

Case VIII.—A male patient, aged forty-nine years, with palpable vessels, had been having a fairly severe epistaxis for four days. He was given a transfusion of two pints of blood on his admission to hospital, the nose was repacked and bleeding stopped; but fresh bleeding occurred every time the pack was removed. The source of hæmorrhage was high up in the nose behind the middle turbinate. This went on for six days, at the end of which time he was given another transfusion and the external carotid was ligated, with complete and immediate cessation of hæmorrhage.

Case XV.—The patient was a male, aged sixty-five years, with much thickened vessels. He had been having a severe epistaxis for some days. On his admission to hospital the nose was packed and he was given three pints of blood over a few days, during which period the nose was repeatedly packed. He was discharged on the seventh day, but six days later was again admitted to hospital, having had a profuse and prolonged epistaxis which reduced his hæmoglobin value to 38%. He was given another transfusion and his nose was packed without effect; then he was taken to the operating theatre and the external carotid artery was ligated. There was still some bleeding from the naso-pharynx after the ligation, and when the middle turbinate was forcibly pushed laterally, examination of the nose showed a feebly spurting spheno-palatine artery on the septum.

A small pack of bismuth, iodoform and paraffin paste was placed over it, and after its removal in a few days' time there was no further bleeding.

Case XVI.—The case of a woman, aged twenty-nine years, illustrates the present routine. She had had a severe epistaxis for three days, and on her admission to hospital her hæmoglobin value was 60%. She was given two pints of blood and her nose was packed; but the hæmorrhage could not be controlled despite reinsertion of the pack on a few occasions. She was then taken to the operating theatre and the external carotid was tied, and she has had no further trouble.

### Comment.

Excluding the first case with Babington's disease, in none of the other eight did I set out to tie the anterior ethmoid. I think the reasons were, first, the difficulty in accurately determining the exact site of the hæmorrhage, particularly when it was far back, secondly, failure to realize the rate at which the ethmoids can bleed, and thirdly, failure to apply the carotid occlusion test accurately. The anæsthetic used was always given by the endotracheal route with a cuffed tube, and induction was by "Pentothal" and a relaxant. As an additional precaution the pharynx was packed.

The external carotid artery was ligated between the lingual artery and the superior thyroid, number 2 chromicized catgut being used for two ligatures. Where there was sufficient length between the ligatures, the artery was severed. A useful means of identification of the external carotid artery, when one is in doubt, is by the aid of the anæsthetist, who can feel the pulsation of the superficial temporal artery and its disappearance on occlusion of the carotid.

In exposure of the anterior ethmoidal artery there is always a fair amount of annoying hæmorrhage. A considerable amount of difficulty may be found in passing a ligature around the artery, and it is much easier and safer to use the diathermy or apply a clip.

### Conclusion.

In conclusion I believe that in the past there has been too much complacency and too much adoption of an attitude of hopeful expectancy in the treatment of these patients with so-called "uncontrolled epistaxis". The sooner the appropriate ligation is performed, the better. I put it forward as a suggestion that if bleeding is not controlled after the patient's admission to hospital, the administration of a blood transfusion, and packing of the nose, the patient should undergo operation.

## Books Received.

*[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]*

"The Year Book of Drug Therapy (1954-1955 Year Book Series)", edited by Harry Beckman, M.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 8" × 5¼", pp. 592, with 74 illustrations. Price: $6.00.

One of the "Practical Medicine Series of Year Books" founded in 1900.

"Handbook of Radiology", edited by Russell H. Morgan, M.D., and Kenneth E. Corrigan, Ph.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 7½" × 5", pp. 528, with 98 text figures. Price: $10.00.

A compilation of quantitative data intended for workers engaged in the clinical, experimental and industrial applications of ionizing radiations.

"The Role of the Pituitary in Cancer: The Clinical Value of Pituitary Lipid Treatment", by Henry K. Wachtel, M.D.; 1954. New York: The William-Frederick Press. 9" × 5¾", pp. 32. Price: $2.00.

The author discusses disturbances of metabolism in cancer, describes experimental studies and reports clinical investigations.

"Manual of Hand Injuries", by H. Minor Nichols, M.D., with a foreword by Michael L. Mason, M.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 9" × 6", pp. 352, with 180 illustrations. Price: $9.50.

Intended for medical students, interns, residents and practising surgeons—"the procedures described are the simplest compatible with good functional results".

"Gynæcology", by Douglas H. Macleod, M.S. (London), F.R.C.P. (London), F.R.C.S. (England), F.R.C.O.G., and Charles D. Read, M.B. (New Zealand), F.R.C.S. (England and Edinburgh), F.R.A.C.S., F.R.C.O.G., with a section on anatomy by James Snyder, M.D., F.R.C.S., and a section on physiology and endocrinology by Russell Fraser, M.D., F.R.C.P.; Fifth Edition; 1955. London: J. and A. Churchill, Limited. 10" × 6½", pp. 876, with 551 illustrations, 27 in colour. Price: 80s.

Based on the principles of "Eden and Lockyer".

"History of the Second World War: United Kingdom Medical Series"; Editor-in-Chief, Arthur S. MacNalty, K.C.B., M.D., F.R.C.P., F.R.C.S. "The Royal Air Force Medical Services", edited by S. C. Rexford-Welch, M.A., M.R.C.S., L.R.C.P., R.A.F. Volume I: Administration; 1954. London: Her Majesty's Stationery Office 10" × 6¼", pp. 634, with 68 illustrations. Price: £3 10s.

The first of three volumes in the series, dealing with the Royal Air Force Medical Services.

"Ciba Foundation Symposium on Chemistry and Biology of Pteridines", edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S.; 1954. London: J. and A. Churchill, Limited. 8¾" × 5½", pp. 440, with 143 illustrations. Price: 42s.

One of the series of "Ciba Foundation Symposia".

"The Casualty Department", by T. G. Lowden, M.A., B.M., B.Ch., F.R.C.S.; 1955. Edinburgh and London: E. and S. Livingstone, Limited. 10" × 7", pp. 286, with 170 illustrations, 12 in colour. Price: 37s. 6d.

The author aims to teach precision—in operating, in diagnosis and in mental processes, all of which he claims are inseparable from good results.

The Medical Journal of Australia

SATURDAY, APRIL 23, 1955.

*All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words of phrases.*

*References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.*

*Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.*

## THE GENETICAL PRICE OF RADIATION.

THE discovery of nuclear fission, leading to the development of atomic energy, has opened up an entirely new source of vast amounts of energy. Unfortunately, in some respects, with the release of atomic energy there are produced tremendous quantities of radioisotopes, which can cause radiation sickness and death in a short time and genetical changes, in the exposed community, over a long time. The production of the radioisotopes is not wholly bad, for they are finding increasing use in biological research and in medical treatment. Controlled, the radioisotopes are extremely useful; uncontrolled, they can do enormous damage, even possibly to the extent of wiping out mankind.

Medical interest in the effects of radiations and radioactivity has been greatly stimulated. On the one side there are the possible deleterious effects on man, whether in the use of atomic weapons in war or in the peace-time applications of the atomic energy production, and on the other is the development of the medical applications of artificial radioactivity. This latter has been so rapid that its impact is being daily increasingly felt by the practitioner of medicine.

The November, 1954, issue of *Medicine Illustrated* is almost wholly taken up by papers by eminent authorities on aspects of medicine in the atomic age. There are papers on the production of radioisotopes and their uses in diagnosis and investigation and the therapeutic uses of radioisotopes. In other articles are discussed the characters and effects of the atomic explosion, the nature and treatment of atomic injuries and the medical organization in civil defence which has been worked out in Great Britain. Because of its general interest and the many misconceptions which have been held regarding the effects, the article entitled "The Genetical Price of Radiation" is particularly timely. This is by Kenneth Mather, Professor of Genetics in the University of Birmingham. The following is a brief summary of Mather's contribution. Genetic materials—genes and chromosomes into which they are organized—are distinguished among the contents of the cell by their permanence and constancy. But they are not immutable; changes in the genes and in the chromosomes, though rare, do occur, and the changed genes and chromosomes reproduce with the same constancy as did their predecessors. Ionizing radiations—X rays, $\gamma$ rays, $\beta$ rays, $\alpha$ particles and neutrons—as well as certain chemicals have the property of increasing the frequency of these changes in genetic materials. The increasing use of radiation in medicine and industry, to say nothing of possible war-time production of vast amounts of radiation, raises the question of what we are doing to man and in particular to his posterity. The only types of nuclear changes which need be discussed are changes in the structure of the chromosomes and mutations in the genes of the reproductive cells. Structural changes arise from breakage of the chromosomes which may be followed by reunion in new ways. As a result of these changes daughter cells may be produced which may be short of genetic material and so die or the nuclei may divide normally but produce offspring which are monstrous or with varying degrees of abnormality. In general these changes lead to some degree of sterility. Gene mutations have no mechanical consequences at cell division, but affect only development and will therefore be passed on to posterity according to mendelian rules. Changes in the chromosomes most often produce partial sterility or changes in the offspring which are very rapidly eliminated by natural selection. The action of selection may operate less directly in man because of his interference with reproduction and survival, but even here there is little doubt that the changes will be eliminated moderately rapidly. Mutated genes are much more important if one takes the long view. They may affect any character of the individual, anatomical, physiological or mental. The effects may be great enough to bring about very early death or so small as not to be noticeable. If the effect of the altered gene is to cause early death or even some reduction in productivity, selection will reduce its frequency and finally eliminate it. The vast majority of mutations are deleterious and undesirable. To increase the mutation rate in man would be to increase the amount of unfitness in the species. Mutant genes are not eliminated immediately, and their spread and persistence in the population and the length of time of their persistence depend on the size of their effects on the individuals carrying them. Most mutant genes are recessive in that, while displaying their effects when homozygous, that is in individuals carrying them in a double dose, they have no effect, or only a very small one, when heterozygous. Even a lethal gene can persist in the population until a male and a female, each heterozygous for it, mate and produce homozygous offspring. It may take 30 generations or more, say nearly 1000 years in man, before the effects will be seen. People may be dying today because of mutations which occurred before the Norman Conquest. But once a mutation has arisen its effect will be seen sooner or later, that is, of course, providing the person harbouring the mutant produces offspring. A point of interest here is the effect of the workings of the welfare State on the disappearance of

deleterious mutants and consequently the general level of fitness of the whole community. Communal action shelters affected individuals and so reduces the effects of a genetically determined disability; the gene or genes in question will tend to spread more widely and there will be more individuals in the community with the disability. Every feature of an individual plant or animal or man is under genetical control; all of them show the effect of gene mutation. It has been calculated that between one in two and one in ten of human germ cells carry a newly arisen mutant gene and that each of us carries at least eight and possibly scores of genes which produce a real, though often slight, detrimental effect.

Now what will be the effect of exposure to ionizing radiations on mutations? Radiations will affect the genes and chromosomes of somatic cells, and so many somatic cells may be affected that the health and even the life of the irradiated individual will be endangered. This, of course, could lead to a marked reduction in reproduction, but the offspring would not be affected. Damage to the germ cells will, however, show in later generations. What could be the magnitude of these effects? Radiation produces all the changes which occur spontaneously. Different doses of radiation and even different types of radiation produce variations in the frequencies with different types of genetic change, so it is not possible to give a simple, accurate answer to the question. However, data are available to allow one to give an approximate answer. All types of genetic change vary in frequency directly with the dose when neutrons or $\alpha$ particles are the agency. Gene mutations vary in the same way with X rays and $\gamma$ rays. If we know the value of $x$, the dose of irradiation which causes as many mutations as occur spontaneously, we can estimate approximately the consequences of any dose $y$ that may be applied to the population. The dose must be applied to the germ line prior to or during the effective period of reproduction. It need not be applied all at once. A very significant fact is that treatments, even the smallest, are cumulative. No matter how the dose may be fractionated and no matter how long is the interval between doses, the genetic effect depends only on the total dose received during the reproductively effective lifetime. Any dose, once given, has a permanent effect. Since the effects are not usually shown for several generations, it is populations rather than individuals that we must consider.

If we set the population at 50 millions, a dose of 50,000,000 $X$ applied to a generation will double the mutation rate and hence, in the long run, will double the genetical disability in the community. If we take thirty years as the average reproductive period the doubling dose may be given over thirty years. That means that something like 1,700,000 $X$ given annually will double the mutation rate. The doubling dose is not known for man, but for *Drosophila* it is of the order of 50$r$ units of X radiation, and there is reason to believe that it is about the same in man. It is improbable that the radiation reaching the gonads of any population at present is anything like the doubling dose, but how much bigger must the dose be to produce seriously harmful effects in future generations? We do not know. The effect of the radiations in man will not become evident for many generations and several centuries. We shall not see the consequences nor will our grandchildren nor even their grandchildren. We have the health of our posterity in our hands now. What are we going to do about it? If we are to consider them, certain precautions are necessary now. The genital organs of people not past the reproductive age should not be exposed to any form of radiation without particularly good reasons, and then only the smallest effective dose should be given. It is to be remembered, too, that the effects are cumulative, and several small doses will have the same effect as one large dose. In the past people working with radiant energy have had the genitals protected to prevent sterility, but doses smaller than those which will produce sterility will produce genetic changes if they are repeated. If, unfortunately, atomic and thermonuclear weapons are used in warfare in the future, the effect on future generations may be extremely serious. Of course, in that case there may be no future generations. The general conclusions to be drawn from this important paper are that radiation effects are cumulative and that a long time must elapse before effects can be apparent.

# Current Comment.

## COMBINED THERAPY FOR AMŒBIASIS.

It is a pity that emetine has such peculiar toxic effects. It is a most rapid and effective amœbicide, yet not only is it being pushed aside in favour of less effective drugs, but it has been turned into a sort of bogy. Granted that emetine alone is not a panacea for amœbiasis, and that for this disease combined therapy must always be used—nevertheless, the tendency to exclude emetine from all systems of treatment instead of making every effort to include it is to be deplored. Henry E. Hamilton[1] in "Treatment of Amebiasis" deals very soundly with the two basic aspects of amœbiasis, and then, frightened off by the bogy of emetine toxicity, finishes with only half a solution to his problem. Hamilton points out that the many variable factors in amœbiasis make it impossible even to lay down criteria of cure, let alone to devise a simple regimen for treatment. The pathogenicity of strains of *Entamœba histolytica* varies; repeated infections occur, often with different strains; host tissue resistance varies, possibly in relation to the state of nutrition of the host; and variations in the intestinal flora affect the nutrition of the amœbæ located in the bowel. In addition, there are two distinct phases of infection: the superficial bowel phase and the deep tissue phase; and the available drugs can be divided roughly into two corresponding groups depending largely on their solubility. Hamilton warns his readers that the superficial and deep phases are always present together, though the many variables may be responsible for their not always being apparent, and he insists that rational treatment must always include drugs aimed at both phases. Even then, he points out, repeated courses of varying drugs will often be necessary for permanent cure. This is so, but he does not discuss this aspect very fully. The pathogenicity of a given strain, and especially its tissue-invading power, vary from time to time. It is not unlikely that during a quiescent interval the amœbæ do not imbibe very much of the circulating drug and so are not destroyed by it—it is even possible that certain drugs at their first impact tend to induce in the amœbæ a stage of reduced activity, thereby producing a temporary cure but leaving the way open for a relapse. Hamilton lists three drugs which are primarily effective against the deep tissue phase of amœbiasis. Of these, "Quinacrine" produces its effect only when it has saturated all the tissues including the skin, which becomes a bright yellow—

[1] *Arch. Int. Med.*, October, 1954.

this takes about five days; in thus saturating all the tissues it is superior to chloroquine, which is selectively stored by the liver; at the recommended dosage of 400 milligrammes daily for fifteen days the risk of mepacrine dermatitis and of mepacrine psychosis is high. Chloroquine is stated to be the drug of choice for "extra-intestinal" amœbiasis, but this is going too far, for that would include metastatic abscesses of bone and other organs, and chloroquine is fairly rapidly taken up from the circulation and concentrated in the liver—that is its great advantage in the treatment of hepatic amœbiasis; it is doubtful whether it reaches a sufficiently high concentration in other tissues to kill amœbæ. Emetine has cumulative effects; it depresses the myocardium to a stage at which even moderate activity can cause permanent damage; moreover, if it is continued in full doses for more than twelve days, the patient may develop an acute confusional psychosis. Nevertheless, it is the most rapid and effective of the amœbicides, even though Hamilton declares, categorically, that chloroquine is the drug of choice—and if Hamilton's opinion is based solely on the results of using the dosage of emetine he recommends (65 milligrammes injected subcutaneously daily for seven days), then his opinion is not really so valuable. A large adult with a healthy myocardium may be given 65 milligrammes daily for twelve days, provided he is kept absolutely at rest in bed for that time and half as long again, and provided a close watch is kept on the state of his heart muscle. This dosage is not to be recommended, however; it is the absolute permissible maximum. An average adult course, perfectly safe, is 65 milligrammes daily for four days, then 33 milligrammes daily for eight days more, with rest in bed and a watch on the heart. For hepatic amœbiasis, this course should have combined with it chloroquine, one gramme daily for three days and then 750 milligrammes daily for two to three weeks. For metastatic extrahepatic abscesses "Quinacrine" may be used instead of chloroquine in a dosage of 300 milligrammes daily for five days, then 200 milligrammes daily for five days, and a suitable antibiotic may also be brought into the combination. For the bowel phase of amœbiasis there is a much wider range of drugs available; but once again it must be emphasized that in association with any superficial ulceration of the mucosa there is always an extensive and potentially much more dangerous deep infiltration of the submucosa. Moreover, the superficially affected areas will certainly have been secondarily infected and perhaps some of the secondary invaders may be more immediately dangerous than the original amœbæ; they may even, by reason of having altered the environmental flora, have driven all amœbæ from the surfaces. Emetine is needed here to attack the deep tissue phase of the amœbic infection, but it has little effect on the superficial bowel phase and none at all on the secondary and commensal invaders, and may even irritate the ulcerated surfaces. Emetine and bismuth iodide, 130 milligrammes given at bedtime in an enteric-coated capsule for twelve days, is perhaps the more satisfactory form in which to administer the drug in these circumstances. Hamilton lists the available drugs for treating this phase as, first, the iodoquinoline compounds, with low toxicity, of which he prefers diiodohydroxyquinoline, 650 milligrammes three times a day for fifteen to twenty days, and second, the pentavalent arsenicals, with more severe toxic reactions, of which he prefers carbarsone. On the other hand Inder Singh,[1] in "The Treatment of Chronic Amœbic Dysentery with Antibiotics in Combination with Other Drugs", states that "Milibis" is preferable because of its low solubility, and finally recommends certain antibiotics, of which oxytetracycline (500 milligrammes every six hours for ten days) and fumagillin (20 milligrammes three times a day for ten days) are the most effective. A triple combination with diiodohydroxyquinoline or carbarsone as the main amœbicide, and oxytetracyline and a short course of emetine as accessories, should prove to be satisfactory; retention enemas of chiniofon or carbarsone should seldom be needed. In every case a modified follow-up course should be given after six weeks, and again after twelve weeks if possible. These recommendations go much further than do Hamilton's, but they are arrived at as a result of following Hamilton's basic ideas right through to their logical conclusion. R. Cattan,[2] in *"Le traitement actuel de l'amibiase intestinale"*, states emphatically that the dangers of emetine have been exaggerated, and that so long as normal precautions are taken there is nothing alarming about the drug. He considers that for acute intestinal amœbiasis emetine has an unequalled value, whereas for chronic intestinal amœbiasis arsenicals and iodine derivatives of oxyquinoline take first place. Dealing with the dysenteric phase of amœbiasis alone, Inder Singh reported permanent cure of 32 patients suffering from chronic amœbic dysentery with a combined course of "Aureomycin", carbarsone, diiodohydroxyquinoline and chloroquine for six days, followed without a break by the last two for fourteen days, although all these patients had previously had single courses of various drugs without beneficial effect. This offers further support for the case for combined therapy, should such a strong case need support.

[1] *Lancet*, March 12, 1955.

## CONGENITAL MEGACOLON (HIRSCHSPRUNG'S DISEASE).

IN a previous reference in these columns to Hirschsprung's disease,[3] the important contribution of Orvar Swenson to its understanding and treatment was described. Swenson has shown that there is a narrowed segment somewhere in the region of the pelvic colon, that the bowel here is deficient in ganglion cells, that this defect is congenital and the dilatation of the colon secondary to it, that by proper radiographic technique this narrowed segment can be demonstrated, and that resection of this narrowed segment is a safe and effective means of curing the disease.

It is amply clear, of course, that with this better understanding and effective treatment of Hirschsprung's disease, it should not be confused with other conditions causing a dilated colon and that diagnosis must be made with care. Great importance attaches to radiographic investigation, which has been discussed in detail by George P. Keefer and John F. Mokrohisky.[3] They stress the need to reserve the term Hirschsprung's disease for those cases in which there is a functional obstruction of the bowel as a result of narrowing of a segment due to an absence (or diminution in number) of ganglion cells in the myenteric plexus. They state that the narrowed segment of bowel may be short or long. In approximately 90% of the cases short segments are seen and in 10% long segments. The typical and most common form occurs in the recto-sigmoid area. The narrowed or spastic segment of bowel usually represents only a part of the aganglionic area, since a rectum also devoid of ganglion cells may appear normal on barium enema examination. In some cases narrowing may be seen of the entire rectum and recto-sigmoid area, corresponding to the entire extent of aganglionic bowel. Not infrequently one finds the aganglionic segment and the narrowed area in the upper or lower part of the rectum. Aganglionic areas in the lower part of the rectum that have been present for some time may not show the narrowed segment, since persistently increased intraluminal pressure may cause this area to dilate down to the anus. The diagnosis in this type of case is difficult, if not impossible, but fortunately the constipation may be controlled by conservative measures. Undoubtedly the true nature of some of these cases is not recognized, and they are labelled as idiopathic megacolon. In the long-segment variety the aganglionic segment most commonly extends from the splenic flexure to the anus. This type can be easily missed if one does not consider the possibility of a long aganglionic segment, or if the barium enema study is limited to the rectum or to the recto-sigmoid area. During the barium enema examination, the rectum, recto-sigmoid area and descending colon may

[1] *Rev. Praticien* (Paris), September, 1954.
[2] M. J. AUSTRALIA, October 31, 1953.
[3] *Radiology*, August, 1954.

appear normal in calibre; so that until the transitional zone between the dilated and narrowed segment is reached, the diagnosis is not apparent, and the findings are therefore interpreted as normal.

Keefer and Mokrohisky go on to state that the disturbance in normal defæcation associated with Hirschsprung's disease is due not only to obstruction by the narrowed segment, but also to loss of the normal defæcation reflex. Fæces pass into the aganglionic segment merely by the propulsive action of the hypertrophied bowel, moving the fæcal column into and beyond the area of narrowing. Once the fæces have passed into the aganglionic segment, the absence of the defæcation reflex and the uncoordinated rectal contractions prevent normal evacuation. It is through further vigorous contractions of the hypertrophied bowel, along with the voluntary relaxation of the external sphincter, that fæces are expelled. Therefore, if compensatory dilatation and hypertrophy cannot overcome the obstruction, further evacuation is impossible, and other measures for emptying the colon must be used. In summary, then, we are dealing with a physiologically abnormal segment of colon, preventing adequate delivery of fæces into a rectum which already lacks propulsive activity.

Describing the radiographic diagnosis of Hirschsprung's disease, Keefer and Mokrohisky state that it is made by demonstrating a narrowed segment of colon, usually in the recto-sigmoid area, with a striking dilatation proximal to this narrowed segment, or so-called "spastic" area. If a narrowed segment is observed, the flow of barium is allowed to enter the dilated bowel for only a short distance, sufficient to demonstrate the extent of the transitional zone—that is, the change from small to large calibre. It is not recommended that complete filling of the colon be carried out when a lesion is demonstrated, as barium impaction or water intoxication may result. In addition, normal saline should be used in preparing the barium suspension mixture to avoid the possibility of water intoxication.

## MALARIA: MISSED DIAGNOSES.

In these columns[1] attention was recently drawn to the incidence of falciparum malaria in Australia, and it was suggested that failure to recognize this condition was due to failure to include malaria in the differential diagnosis, largely because of failure to take full case histories. Now, Louis A. Hall,[2] in "Korean Vivax Malaria", discusses the occurrence of vivax fever in the United States of America among ex-servicemen from Korea. This article contains much that could be taken to heart in Australia. *Plasmodium vivax* is not a vicious killer like *P. falciparum*, but it can cause much unnecessary debility, and it is not so easily controlled. Unlike *P. falciparum*, it goes through a series of secondary exo-erythrocytic stages, and the only drugs which will eliminate these stages are the 8-aminoquinolines, of which even the safest, primaquine, is too poisonous for unsupervised routine use. The other available drugs are suppressant only, and after discontinuing them, persons harbouring latent vivax or malariæ infections will sooner or later develop a series of attacks of malarial fever; and it is hardly practicable to put every person who has had a sojourn in a malarious country to bed and give him a carefully supervised fourteen-day course of primaquine. Hall is concerned only with the Korean strains of *P. vivax* with their latent period of nine or ten months, followed by a series of short-term relapses; in Australia the strains customarily met with have been those from New Guinea, with a short relapse term of about six weeks—differences derived from the absence of any cold season in New Guinea on the one hand, and the occurrence of a severe winter in Korea on the other. But the fact that Australia now has ex-servicemen of her own who have served in Korea makes it likely that there will be a double problem here. Hall states that in the U.S. Naval Hospital at Oakland, up to 50% of attacks of vivax fever were initially diagnosed incorrectly, the percentage of missed diagnoses increasing year by year. He considers that the main factors responsible for mistaken diagnoses were the patients' histories; the long latent period after the patients had returned to their country, lengthened perhaps by the taking of suppressive drugs for some time after their return, let the history of residence in Korea slip into the background. Yet malaria is so protean in its manifestations, and at the same time presents so few outstanding features likely to suggest the correct diagnosis to anybody not reasonably familiar with it, or not on the lookout for it, that eliciting the significant feature in the patient's history may well be the only guide to correct diagnosis. In Australia, with two strains of *P. vivax* to be considered, the vast differences between their relapse times may still further complicate the problem. It is just as well that *P. vivax* is not a rapid killer, but affords time for the patient or his attendant to delve further into the history and come up with a lead to the correct diagnosis before it is too late.

[1] M. J. AUSTRALIA, April 2, 1955.
[2] *U.S.A.F. Med. J.*, January, 1955.

## SOME ASPECTS OF ASTHENIA IN DISPLACED PERSONS.

Most people in this country must by now have had some contact with New Australians who have in past years been displaced persons. Certainly, owing to the careful selection of migrants, the majority are of a high standard of physical and mental fitness. Some concessions have been made, as was pointed out by I. R. Mackay[1] in this journal, in the following words:

> Admittedly, some substandard individuals were probably accepted, and although the primary objective was the recruiting of suitable migrants, a substantial contribution to the solution of the European resettlement problem could reasonably be said to have been made. Finally, the knowledge of how much it meant to these unfortunate victims of circumstance to leave behind them a miserable and aimless existence might have sometimes influenced medical decisions in their favour.

In some cases the assimilation process has been difficult both for the migrants and for their new countrymen; in others the process has been relatively smooth. Any observations which can help us to a greater understanding of people who have suffered from being held in concentration camps, with all their terrible implications, should be carefully considered and made widely known. For this reason some facts brought to light in two international congresses on the pathology of displaced persons held during 1954 are of interest. The first was held at Copenhagen in June, the second at Paris in October; A. Bacharach[2] gives an account of these congresses particularly from the point of view of the strange "fatigue" that continues to plague these victims of man's inhumanity to man. Sufficient is known, even in this land distant from the scenes of horror and after this lapse of time, to make it unnecessary to recount again the details of the cruelty and deprivation to which they were exposed. Bacharach's figures refer solely to French displaced persons; these numbered 230,000, and of them 28,000 escaped with their lives and 15,000 have died since their return home.

> It is obvious that all these repatriated people bear the stigmata of transportation. In the nine years that have elapsed we have become convinced that no human constitution whether male or female, no matter how outstandingly strong it may have been before imprisonment, could remain unaffected by the physiological distress and psychological damage resulting from transportation.

Bacharach is concerned that the special type of asthenia affecting repatriated displaced persons should be understood. He points out that elimination of any discoverable ætiological factor should be undertaken by means of all

[1] "Medical Selection of Migrants from the Displaced Person Population of Post-War Europe", M. J. AUSTRALIA, March 14, 1953.
[2] *Presse méd.*, January 15, 1955.

possible clinical and laboratory investigations; that is mandatory. Progressive cachexia always brings about asthenia in the sense of general fatigue; but in the great majority of persons in this series it has persisted for as long as nine years after their repatriation. Of 57 subjects examined in 1952, 31 (just over half) presented this type of asthenia; three of them had hypotension without any other physical abnormality. In January, 1953, examination of another group of subjects showed that approximately half had asthenia of various types. Bacharach believes that the majority of displaced persons are asthenic; the asthenia may accompany an organic lesion, or it may be present without any detectable organic lesion, or it may be of psychological or intellectual type.

With regard to the first type, symptomatic asthenia, it is obvious that the cause must be sought by every possible means; the repatriated subject is not exempt from any known pathological condition. It is also obvious that when the cause is found it must be treated, and that is all that need be said about symptomatic asthenia. The second type, asthenia resulting from hunger, was the commonest type in all concentration camps. It is characterized by progressive muscular weakness in the arms, hands, legs and trunk, by slow and awkward movements during work, by difficulty in standing upright and in running, and by increasing weakness on the slightest exertion. If repatriation and the resumption of normal life have succeeded in restoring the basal metabolic rate, the subcutaneous and orbital fat, the depleted muscles and the respiratory and cardiac functions, and in giving a normal appearance to the prematurely aged human being, it remains none the less true that physical asthenia persists in a large number of displaced persons and internees, especially after a long and painful period of detention in particularly cruel concentration camps. This somatic asthenia, or myasthenia, is the true asthenia without discoverable organic cause found among displaced persons. It is characterized by great physical fatigability which prohibits all exertion. The patient needs a will of iron if he has to undertake a difficult task or walk any distance. He does not waste movements, and prefers to remain still, or to sit or lie down. The syndrome resembles true essential muscular hypotonia or myasthenia. It is chiefly localized to the inferior extremities, and is often accompanied by joint pains, particularly in the ankles, without radiological signs. Sometimes *meralgia paræsthetica* is also present, with sensitivity to cold but no definite organic vascular disorder. This type of asthenia persists long after the avitaminosis, loss of weight, nutritional œdema and all the sequelæ of malnutrition have become only a distant memory. The third type of asthenia is late intellectual asthenia. Bacharach describes it as a cerebral fatigability manifested by "emptiness in the head" not induced by brain work, impaired memory, confusion of ideas, and inability to follow a lecture. Other characteristics of the condition are slowness of reactions, and lack of interest (even in a normally intelligent person) in investigating a problem or in turning it to his professional advantage. The patient shrinks from making the slightest effort, and even from taking the steps necessary to obtain a pension or reparations for his possessions lost during the war. He shows inertia in his relations with the administrative or military authorities, and has to be directed and guided like a child. He knows exactly what he has to do to get out of his difficulties, but his only reaction is complete inertia. Psychic or emotional asthenia is often the corollary of somatic asthenia. The patient suffering from psychic or emotional asthenia presents apathy alternating with great irritability; he has difficulty in readjusting himself to his social or family life. Genuine "incompatibility of temperament" has been the cause of the separation or divorce of husbands and wives who had lived in complete harmony before the upheaval. Bacharach remarks that he has observed this disordered temperament much more frequently in men than in women. During periods of exacerbation the subject may show neurotic phenomena arousing suspicion of schizophrenia or hyperthymia. The condition is nothing more than psychasthenia which has become chronic, with simple functional sequelæ and no mental illness. The attacks are paroxysmal, and are often accompanied by tremors, flushing of the face and headache. Usually it is not the patient but his relations who reveal their occurrence and their severity.

In another investigation, A. A. Hulshoff[1] has given an account of the medical examination of repatriated Dutch nationals from Indonesia in the years between 1946 and 1951. He also has something to say about "fatigue", which was found to be one of the most frequent complaints, and often the principal one. Hulshoff and his colleagues tried to discover its causes, and to this end they divided their clinical material into the following two groups: (i) 1514 ex-internees who had suffered from malnutrition; of these 484 (32%) complained of fatigue; (ii) 153 ex-servicemen who had been well fed all along; of these 38 (25%) complained of fatigue. Hulshoff points out that the difference is not significant. The patients complaining of fatigue were then grouped according to the diseases from which they had previously suffered, so that a crude comparison was possible. It was found that fatigue was an equally frequent sequel of tropical and of general diseases, but occurred with much greater frequency after deficiency diseases. In 11 of the internees liver disease was the only possible organic cause; but none of the ex-servicemen who complained of fatigue had liver disease, and they had suffered from malaria and dysentery as severely as the ex-internees. In the discussion on Hulshoff's paper, Van Wulfften Palthe said that the almost complete absence of complaints of fatigue during the period in camp (except towards the end) was not due to insufficient inquiry on the subjects' return to the Netherlands, or to failure to remember the fatigue because it was superseded by more serious physical symptoms and psychic experiences. In the camps of Batavia at least, fatigue was not a general symptom as long as nutrition was not extremely poor. White people in the tropics fairly commonly suffered from leiodystonia, of which fatigue was a characteristic sign. The fatigue was paradoxical, in that it was made worse by rest and disappeared on physical exertion and vigorous mental activity. He thought that, in the camps of Batavia, the physical exertion, the wearing of a minimum of clothes and the strong psychical stimulus of resistance to the oppressor brought about an improvement in adaptation, so that fatigue did not occur. Towards the end when the food became poor, the syndrome of exhaustion appeared; subjectively it was felt, not as fatigue, but as apathy. At the same time the complaint of sacro-iliac pain became common; the speaker considered it to be due to stretching of bands (the muscles were flaccid because of protein deficiency, and all fat had disappeared), salt depletion and disturbances of calcium metabolism (many people became shorter by several centimetres). Other points in the discussion relating to fatigue need not be mentioned in this place. In any event, Hulshoff's paper on the diseases affecting these Dutch nationals is rather in the nature of a preliminary communication, and further work is to be done. With further reference to the effects of malnutrition, attention may be drawn to an observation made by A. A. J. van Egmond, J. J. Groen and G. de Wit[2] in the course of some work designed to aid in the selection of individuals susceptible to motion sickness. They found that a tendency to seasickness in some persons, chiefly ex-prisoners-of-war, could be explained only on the basis of an irreversible lesion brought about by vitamin *B* deficiency, involving especially the pellagra-preventing factor.

It is evident that investigations on these two groups of nationals, whose experiences were not strictly similar in every way, have produced slightly different findings. What does seem to be established is the existence of the syndrome of asthenia of displaced persons—Bacharach is convinced of its reality. If this is so, then every effort should be made to understand it, for the unfortunate sufferer may well present a highly uncomfortable diminished tolerance to life, affecting his health and his social, family and professional relationships. It is to be hoped that further investigations into the problem will be undertaken.

[1] *Acta leidensia scholæ med. trop.*, Volume XXIV, 1954.
[2] *Internat. Rec. Med.*, December, 1954.

# Abstracts from Medical Literature.

## BACTERIOLOGY AND IMMUNOLOGY.

### Non-Infectious Influenza Virus.

H. S. GINSBERG (*J. Exper. Med.*, December, 1954) studied the formation of non-infectious influenza virus in mouse lungs, and showed its dependence upon extensive pulmonary consolidation initiated by the viral inoculum. He inoculated mice intranasally with a mixture of living and heated allantoic fluid of a strain known to produce pulmonary consolidation, in a proportion of 99·9% non-infective particles and 0·1% infectious particles. There was extensive pulmonary consolidation and development of high-titre agglutinins, but no increase in infectious titre. The non-infectious material developed during the initial cycle of virus reproduction, and was demonstrated as hæmagglutinin; it could not be progagated, and only fully infectious virus could be demonstrated in secondary passage. Inoculation of a small amount of the original mixture resulted in a yield of high-titre infectious virus and but little of the hæmagglutinating component, and this was related to a smaller amount of consolidating lesion. The non-infectious form was not produced as a result of the interference phenomenon. The author concludes that non-infectious virus was not immature or incomplete virus, but might be inactivated virus or "some aberrant form of the agent".

### Interference Immunity in Pertussis.

D. G. EVANS AND F. T. PERKINS (*Brit. J. Exper. Path.*, December, 1954) have continued their studies of experimental immunity in pertussis, and demonstrated the mechanism of a previously reported observation. This was that after a single intraperitoneal inoculation of pertussis vaccine, mice developed an early immunity to intracerebral challenge which was independent of circulating antibodies. The present experiments consisted in mixing the vaccine with the challenge dose and inoculating the two intracerebrally, along with a control group of mice given living organisms without vaccine. The control mice uniformly died within seven days; while the mice receiving varying challenge doses and varying dilutions of vaccine always survived longer, some up to twenty-one days, even when the challenge dose was 110 $LD_{50}$. Three different vaccines behaved similarly. The protective effect of the vaccine was also demonstrated when given three hours before the challenge dose. The authors believe that the interference may be similar to that occurring in virus infections, in which the phenomenon of interference is well known.

### Experimental Nephritis.

R. W. REED AND B. H. MATHESON (*J. Infec. Dis.*, September-October, 1954) have studied the effect of a single exposure to type 12 streptococci in the production of experimental nephritis. A localized infection was produced by subcutaneous injection of 0·1 millilitre of culture in the dorsal lumbar region and was terminated after ten days by intramuscular injection of penicillin. Antistreptolysin O titres in the rabbit serum were determined at intervals, twenty-four-hour samples of urine were collected and analysed, and blood pressure readings were taken. Control animals which had been inoculated with sterile culture medium were also observed, and a further experimental group of animals were inoculated intravenously with sterile filtrates from type 12 cultures over a period of eighteen days. The normal control and sterile culture medium groups showed no variation from normal blood pressure readings of 25 to 30 millimetres of mercury. In the group infected with living organisms hypertension developed after three weeks; and in some animals albumin and red cells appeared in the urine, also at periods after three weeks. The animals injected with filtrate intravenously developed hypertension during the period of the injections to a roughly similar degree, and urinary albumin and red cells also developed. The antistreptolysin O titres rose sharply in the animals infected with living organisms. Post-mortem examinations of the kidneys of the animals showed healing lesions of lower nephron changes and some cellular infiltration of glomeruli. The authors found these changes more consistently in the experiments in which they used type 12 streptococci than in those in which other types were used, and they believe that there is some special nephritogenic agent released in the growth of this type of streptococcus.

### Viræmia in Experimental Poliomyelitis.

D. BODIAN (*Am. J. Hyg.*, November, 1954) studied general aspects of infection after intravascular inoculation with strains of high and low invasiveness. Using cynomolgous monkeys and inoculating three types of virus by cardiac puncture, in one-millilitre quantities, he gathered evidence that direct invasion of the central nervous system could occur through the bloodstream. Virus titrations and neutralization tests were carried out in tissue cultures, and for histological studies tissues were taken from *medulla oblongata* (*area postrema*) and from cervical and lumbar enlargements. A preliminary experiment showed that the insertion of a virus-contaminated needle through nerve fibres was not sufficient to initiate infection, and careful histological examination failed to show evidence that virus had been excreted from the blood-stream to the olfactory or trigeminal nerve routes. However, it was soon apparent that in animals in which lesions in the central nervous system appeared suddenly, usually in one arm, there was a suggestion that virus had penetrated at a single point, and histological examination showed this to be in the *area postrema* in a proportion of cases, or in the cervical cord. With the Mahony strain (type 1) the attack rate after intracardial inoculation was never greater than 50%, while types 2 and 3 succeeded in paralysing only an occasional monkey; so that paralytic titres for these viruses could not be tested by this method. Antibody response could be shown in all animals in which viræmia developed; but in those in which virus could not be recovered from the blood, no antibodies appeared. The incubation period of the disease after intravascular inoculation was similar to that after intracerebral inoculation, but shorter than that after virus feeding.

### Metabolic Aspects of Host-Parasite Interaction.

L. J. BERRY AND R. B. MITCHELL (*J. Infect. Dis.*, November-December, 1954) studied some metabolic aspects of host-parasite interaction, using the albino mouse and *Salmonella typhimurium*. They state that when mice are inoculated intraperitoneally with about 250,000 bacteria, the infection can be followed by counts of organisms in the blood, where there is a steady increase up to ten hours, then a marked drop to twenty-four hours, and then an exponential increase up to the time of death about seventy-two hours after inoculation. The blood sugar level in such mice may be up to 163 milligrammes *per centum* at the beginning of the experiment, but falls to a low level and remains there from seventeen hours until death; this also follows inoculation of heat-killed organisms, and may be attributed to endotoxins. If such mice are also given a series of injections of sodium malonate 20 milligrammes in 0·5 millilitre of normal saline, the resistance to infection is lowered, the counts of organisms in the blood have risen to a maximum in twelve hours, and the survival time of the animals is shortened to twelve to twenty-four hours. Similar results can be obtained with succinate, and after two injections of fluoroacetate; while mice given citrate behaved comparably to uninjected mice. There can be shown a decrease in lactic acid in the heart, kidney, duodenum and, to a less extent, liver, in mice given heat-killed organisms. The authors discuss the part played on the injection by inhibitors of the Krebs cycle, such as malonate, the part played by the infection on the blood sugar level, and the disturbances of carbohydrate utilization shown by the decrease in lactic acid. They suggest that the biochemical effects of pathogenic bacteria and the mode of action of Krebs cycle inhibitors may be studied in this type of experimental approach.

## HYGIENE.

### Evaluation of a Health Education Programme.

A. L. KNUTSON AND B. SHINBERG (*Am. J. Pub. Health*, January, 1955) discuss the need for and methods of evaluating systems of health education. Evaluation is defined as "the process of determining the worth or value of something relative to a given purpose". The authors state that in this sense programme evaluation is a continuous process beginning even before programme plans are formulated. Its purposes are to provide valid estimates of effectiveness in achieving specific objectives and to provide guidance in carrying out programme activities. To achieve both purposes, two types of evaluation are needed: measurement of programme achievement and programme progress. Although either kind of evaluation can be applied independently of the other, both may be applied concurrently, even within the same study. The authors have attempted to illustrate this by discussing a study of the effectiveness of television as used by the National

American Red Cross in teaching home nursing. The findings of this study suggested that the teaching of home nursing by television under the conditions described was in general as effective as classroom teaching. By including in the study of programme achievement some approaches to yield information on programme progress many useful suggestions for programme improvements were obtained.

### Agricultural Chemicals and Public Health.

W. J. HAYES (*Pub. Health Rep.*, October, 1954) discusses the potential hazards and benefits to public health resulting from the manufacture and use of agricultural chemicals. He states that hazards associated with agricultural chemicals may be classified as follows: (i) hazards of manufacture or formulation; (ii) hazards of mixing or application; (iii) hazards to persons not directly associated with the chemicals—namely, (*a*) children and irresponsible adults, (*b*) persons whose food is contaminated by residues of the chemicals, and (*c*) persons exposed incidentally. Reference is made to a number of investigations in which the incidence of disease has been compared in areas where insecticides are little used and in areas where they are extensively used. Comparisons have also been made in some areas of the disease incidence before and after the intensive use of insecticides. Where organic phosphorus insecticides were used, cholinesterase values were estimated for exposed persons. Here extensive day-to-day exposure was regularly associated with reduced blood cholinesterase levels; but when fatal or near fatal poisoning occurred, it always followed brief massive exposure. Slight illnesses due to over-exposure always resembled severe poisoning in a milder and briefer form. No evidence was found that agricultural chemicals used as insecticides were the direct or indirect cause of any chronic disease or a contributing factor in diseases generally recognized as having other ætiologies. Persons engaged in manufacturing and applying insecticides were rarely poisoned if precautions were taken and instructions followed. Proper training and supervision by the employer or foreman were necessary. In the basically agricultural community it was usually found that the necessity for using toxic chemicals was accepted. Each grower took direct personal responsibility for the chemicals used and for the precautions necessary. Neighbours were advised when toxic chemicals were being used and usually recognized the necessity for their use. As a result there was a low incidence of poisoning.

### Sanitation Aspects of "Take Out" Foods.

C. L. SENN AND P. P. LOGAN (*Am. J. Pub. Health*, January, 1955) draw attention to the need for hygienic safeguarding of a new group of prepared foods. These are frozen pre-cooked foods and include meat, poultry and vegetable pies, cooked fish and poultry, and even complete meals. They are kept in "serve-yourself" freezers from which they may "be taken" by customers. The use of these "take out" foods is increasing in homes, in restaurants, and particularly in industrial canteens in the United States of America. The authors consider that in the prevention of enteric disease epidemics due to fresh frozen and pre-cooked frozen foods it should be recognized that (i) various paratyphoid organisms, capable of producing intestinal and other complicating infections in man, have been isolated from eviscerated poultry, (ii) paratyphoid and typhoid bacilli survive for long periods of time in frozen foods, and (iii) the botulism organism and its toxin survive indefinitely in frozen foods and remain potent after prolonged freezing. The authors describe how different processing methods were investigated bacteriologically and give the results obtained. They conclude that "take out" foods should be prepared and processed so that storage at improper temperatures or failure to follow instructions on the labels will not cause illness. The present objective might well be to package these foods at a high enough temperature to destroy non-sporeforming organisms. Future attention may well be directed to some more positive means of making these foods as safe as commercially canned products, such as the use of ionizing radiation to sterilize the product after it is packaged. There is need for further studies in order to ensure that procedures give adequate protection against botulism. There should be public education to allay the unjustified fears of consequences from partial thawing and re-freezing of foods. The public should be advised of those measures which are really important for them to take for their own protection. They should be warned to avoid prolonged holding or storage at temperatures favourable for the growth of organisms. Data on actual illness attributed to "take out" foods should be collected. Further research is necessary to determine practical and efficient methods to protect "take out" foods from contamination.

### Exposure to Safe Solvents.

J. P. HUGHES (*J.A.M.A.*, September 18, 1954) discusses hazardous exposure to so-called safe solvents. He states that solvents are used in nearly all industrial plants. Some supposed safe solvents have a toxic effect. He reports a case of pulmonary œdema caused by methylene chloride (dichloromethane), which is used as a paint remover, refrigerant and degreasing solvent. He states that it is narcotic and has irritating effects on the respiratory system. Tetrachlorethylene is recorded as giving rise to acute hepatitis. Carbon tetrachloride gave rise to severe nausea and vomiting in a girl working with this substance. Methanol, used in the reconditioning of metal drums, caused toxic amblyopia, vomiting and mental confusion, mainly after ingestion by employees in the place of wine.

## MEDICINE.

### Œsophageal Reflux.

B. CREAMER (*Lancet*, February 5, 1955) describes an investigation in which the intraœsophageal and intragastric pressures were measured in an attempt to define the changes accompanying œsophageal reflux and to find if any constant pattern of events precipitated reflux. The pressures were measured through fine "Polythene" tubes with Hansen capacitance manometers. Twelve patients with œsophageal reflux were investigated; in four of these the cardia was in the normal position as determined by barium studies, and in eight a hiatus hernia was present. Twelve subjects with normal function of the cardia were studied as controls. As a result of the study, the author states that œsophageal reflux is accompanied by changes in the intraœsophageal pressures which can be used to detect its occurrence. Reflux takes place only during inspiration and more commonly in certain positions of the patient than in others. There is no appreciable change in the pressure gradient across the cardia when the patient is in these positions and it is postulated that the competence of the cardia depends on its anatomical shape. No evidence was found of reverse peristalsis, and the author states that the observed facts fit well with the conception of a mucosal valve guarding the cardia.

### Aplastic Anæmia following Mepacrine.

M. D. PATON, M. J. RIDDELL AND J. A. STRONG (*Lancet*, February 5, 1955) report a case of fatal aplastic anæmia occurring in a patient with localized *lupus erythematosus* who had been treated with mepacrine. They state that it would seem well to bear in mind the possibility of bone marrow damage in patients treated with mepacrine. Although agranulocytosis can occur without warning, impending marrow aplasia may sometimes be recognized before irreparable damage has occurred if regular blood counts are carried out.

### Dietary and Serum Levels of Cholesterol in Vegetarians.

MERVYN J. HARDINGE AND FREDERICH J. STARE (*J. Clin. Nutrition*, March-April, 1954) discuss the dietary and serum levels of cholesterol, and report on the cholesterol intakes, as determined by calculation and serum cholesterol measurements, of a limited number of "pure" vegetarian, lacto-ovo-vegetarian and non-vegetarian pregnant women, adolescents and adults of both sexes. The dietary cholesterol is higher in amount in the non-vegetarian groups than in the lacto-ovo-vegetarian groups, whereas the "pure" vegetarian diet is cholesterol-free. Serum cholesterol levels of the adult vegetarian groups tend to be lower than those of the adult non-vegetarian groups, the "pure" vegetarians having the lowest values. The significantly lower serum cholesterol concentration of the "pure" vegetarians occurred despite a free intake of vegetable fat. Cholesterol levels appear more closely related to the intake of animal fat than of total fat. The higher serum cholesterol concentrations of the older adult groups, as compared with the younger groups, could support the observation of others that there occurs a gradual increase in cholesterol levels with advancing age. Since the differences in serum cholesterol in the adolescent age groups is only slight, it is suggested that with aging, there may occur a diminishing ability of the body to handle excess cholesterol, whether of endogenous or exogenous origin.

# Medical Societies.

## THE OTO-LARYNGOLOGICAL SOCIETY OF AUSTRALIA.

THE annual general meeting of the Oto-Laryngological Society of Australia was held in Melbourne on August 16 and 17, 1954, DR. B. B. BLOMFIELD, the President, in the chair.

### Election of Office-Bearers.

The following office-bearers were elected: *President*: Dr. B. B. Blomfield. *Vice-President*: Dr. G. C. Halliday. *Secretary-Treasurer*: Dr. V. G. Bulteau. *State representatives*: Queensland, Dr. A. Quayle; New South Wales, Dr. V. G. Bulteau; Victoria, Dr. R. Willis; Tasmania, Dr. G. J. Ramsay; South Australia, Dr. R. von der Borch; Western Australia, Dr. N. M. Cuthbert.

### Three Little Known Aural and Nasal Operations.

DR. A. B. K. WATKINS (Newcastle) read a paper entitled "Three Little Known Aural and Nasal Opeations" (see page 593).

DR. G. C. SCANTLEBURY (Melbourne) said that he had never seen or heard of the operation for *perichondritis auris*. As far as the turbinate operation was concerned, Stewart Lowe had first published details of that operation about 1919, and Dr. Andrew, of Melbourne, and Dr. Scantlebury himself frequently performed it, but he did not take his incision as far along as Dr. Watkins. With regard to the third operation, Dr. Scantlebury said that he had always favoured the Cushing route to the pituitary. Often one found that the tumour had eroded the bone, and it became difficult to define the sphenoid sinus. One tumour he had encountered proved to be a mucocele of the sphenoid. An advantage of the sublabial incision was that one was always working in the mid-line.

DR. R. VON DER BORCH (Adelaide) inquired whether Dr. Watkins had noticed an increase of perichondritis since the endaural aproach to the mastoid had come into general use.

Dr. Watkins, in reply, said that he had not noticed any particular connexion.

### Fenestration Operations.

DR. G. C. HALLIDAY, DR. H. D. RAFFAN and DR. R. G. MACKAY (Sydney) presented a paper entitled "Some Aspects of 500 Fenestration Operations" (see page 597).

DR. S. SUGGIT (Brisbane) read a paper entitled "Closed One-Stage Fenestration" (see page 601).

Dr. Watkins, in referring to the amount of bleeding at fenestration operations, said that he noticed that most surgeons blamed the anæsthetist for it. He had frequently been bothered by bleeding until he noticed that Garnett Passe had very little trouble with it. He found that Passe injected adrenaline in the strength of 20 minims to one ounce of local anæsthetic solution, but he rarely used more than half an ounce of the solution. Dr. Watkins had adopted that strength, and had found that by using it he could almost forget about any trouble with bleeding. If "Pentothal" was used, it was preferable to have it administered by the continuous drip method. With reference to infection, he had found that there had been much less of it since the number of dressings had been limited. Furthermore, he now used tantalum sutures, which could be left in for about ten or fourteen days, and then no more dressings were necessary.

In discussing Dr. Suggit's method, Dr. Watkins said that one should aim at the ideal of closed cavities. However, he wondered whether Dr. Suggit's method would stand the test of time; it seemed risky to put a free skin graft onto a structure like the semicircular canal, where there was a likelihood of sloughing. The graft involved leaving the *stratum corneum* and *stratum lucidum* inside a closed cavity, and it was interesting to speculate whether cholesteatoma might form in years to come.

DR. D. F. O'BRIEN (Melbourne) said that he envied the conditions under which Dr. Halliday was able to operate. Referring to the audiograms showing a high frequency drop, he said that he found that the response to Rinne's test was sometimes negative and sometimes positive, and he often wondered why that should be. In speaking of infections, Dr. O'Brien said that it was his experience that even when infection had occurred it did not seem to have any effect on the flap or the drum. He wondered what packing had been used. In the assessment of results, Dr. O'Brien thought that it was impossible to be completely scientific. For instance, he often found that about six months after operation there was some deterioration, but if the loss at 2048 cycles per second did not exceed 30 decibels the patient was still happy. He had experimented with a platinum-iridium stopple. The result had shown no lift in the lower frequencies, yet the patients were satisfied because the hearing at 2048 cycles per second was still up. He referred to Lindsay's suggestion of putting a cotton plug soaked in oil over the drum, thereby producing a 10 to 15 decibel improvement. Dr. O'Brien said that he had one intelligent patient who was able to do this. Referring to closed fenestration, Dr. O'Brien said that he had tried three operations a few years earlier using Holmgren's method. Two patients quickly returned to their pre-operative level of hearing. On reopening one of the cavities soon after, he had found a soft scar in the window, but no bony overgrowth.

DR. C. C. WARK (Brisbane) asked about the percentage of phenyl mercuric acetate in methyl alcohol. He next inquired whether any explanation could be given why patients should have a pronounced dip for bone conduction, well below air conduction, at higher frequencies. Dr. Wark said that he had performed ten operations by Dr. Suggit's technique, but he had favoured conjunctiva, because there was no keratin in it and it was not likely to form sebaceous secretion. He thought that the indications for operation might be much broader when that technique was used, and instanced the case of a mentally deficient girl living 600 miles away in the outback of Queensland who had no hope of attending for regular dressings. That he thought was a good argument against producing the ordinary radical mastoid cavity. He had had his failures with the closed method; for example, one patient had gone home on the ninth day feeling well. She had travelled in a taxi which went very fast, and that made her extremely giddy. She was readmitted to hospital, where he reopened the cavity, and failed to find adhesions; the flap was quite adherent. Subsequently the patient settled down and became very well. There had been three failures. One of the three patients had had a great deal of hæmorrhage, and he now wore a hearing aid in the treated ear. Another, operated on a month prior to the meeting, suddenly became totally deaf and very giddy on the third day after operation. Dr. Wark said that he had reopened the cavity, but found the flap still in place. However, the labyrinth was still active, because the patient was giddy on blowing her nose.

Dr. von der Borch said that he had performed four closed operations, but he had performed them in two stages. For two he had used conjunctiva, and for the other two Thiersch grafts followed by gold leaf and then "Calgitex". The conjunctiva did not seem to adhere well, but the skin did. He emphasized the importance of pressure, and pointed out that it was possible to remove the "Calgitex" by removing one suture. Dr. von der Borch asked Dr. Halliday whether there was any relation between paracusis and the results obtained by Dr. Raffan and Dr. Mackay. Also he asked what was the effect of fenestration in preventing serious nerve loss.

Dr. Halliday, in reply to Dr. O'Brien, said that he had no answer to his question on the variability of the response to Rinne's test. As far as sloughing was concerned, in two cases there had been nothing of the drum or the flap left at all; yet he had convinced himself at operation that it had been a good flap. The packing he used was gauze soaked in Ringer's solution in the cavity and "Porowax" over the window. With reference to the stopple, Dr. Halliday said that it had been almost universally abandoned. The strength of phenyl mercuric acetate recommended was one in one thousand. In reply to Dr. Wark's second question, Dr. Halliday said that he had no explanation for the dip in bone conduction at high frequencies. In reply to Dr. von der Borch's question about paracusis, Dr. Halliday said that of the first 50 patients 44 had had it, and of the second 50, 37. In Dr. Raffan's series, 77 out of 86 patients had had it, and in Dr. Mackay's series, 28 out of 33. In other words, there was not much difference in the three series.

Dr. Suggit, in reply, said that Dr. Watkins had brought out doubts which had been in his own mind for over a year before he had tried the closed method. Referring to the fears about the formation of cholesteatoma, he said that he thought the outside provided a good safety valve. Dr. Suggit himself did not consider that skin provided the best closure, and thought it would be better if something else could be devised. Referring to "Calgitex", he said that in

his opinion it should not be left in for more than eight days. Speaking of vertigo, Dr. Suggit said that he had seen the same thing in two patients who had undergone the classical operation. Referring again to packing, Dr. Suggit proposed using gelatine sponge in the future.

Neither Dr. Halliday nor Dr. Suggit knew the answer to Dr. von der Borch's question about the prevention of serious nerve loss, nor could anyone know the long-term results.

### Ménière's Syndrome.

DR. C. C. WARK (Brisbane) read a paper entitled "Some Aspects of Those Conditions Provisionally Diagnosed as Ménière's Syndrome" (see page 603).

DR. A. SCHWIEGER (Melbourne) read a paper entitled "Some Aspects of Those Conditions Provisionally Diagnosed as Ménière's Syndrome" (see page 605).

DR. R. BLAUBAUM (Melbourne) discussed a patient who had been well controlled by nicotinic acid for three years, but suddenly found her condition aggravated by it. Dr. Blaubaum stopped it and administered "Antistine" intravenously, which controlled the giddiness; so the patient continued to take "Antistine" by mouth until it subsequently aggravated the giddiness, when Dr. Blaubaum again switched back to nicotinic acid. He asked whether any explanation for the occurrence could be given.

DR. A. S. DE B. COCKS (Adelaide) said that the definition of vertigo did not seem to him to matter, because it was not what the surgeon thought but what the patient thought that mattered. For that reason it was the history that was important. The finding of labyrinthic hydrops Dr. Cocks thought was only an effect, and there must obviously be something behind it. The finding of loudness recruitment was important. He had treated three patients by sympathectomy; two had been relieved, but one had developed ataxia and he was inclined to wonder whether the diagnosis was correct. He had been waiting for an opportunity to give an injection of local anæsthetic agent into the stellate ganglion during an attack, but it had not yet arisen.

Dr. Halliday, referring first to unconsciousness arising from giddiness, said that he did not believe that that was so, having been taught that it was a diagnostic point that true giddiness did not go on to unconsciousness. Secondly, he supported the statement about the importance of recruitment, and referred to an investigation in Sweden which showed that eighth nerve tumour was the third most common intracranial tumour; most of 300 patients with that condition had been examined by ear, nose and throat surgeons, and the disorder had been dismissed as of no consequence. The moral was that patients with unexplained unilateral nerve deafness should be kept under observation. Thirdly, Dr. Halliday asked whether Dr. Wark had had any experience of treatment by injections of streptomycin.

DR. D. F. COSSAR (Melbourne) referred to the contention that postural nystagmus was a benign condition. He asked, if that was so, what the long-range prognosis was.

DR. B. HILLER (Hobart) said that if the statement that a patient suffering from true Ménière's syndrome could become unconscious was true, he would like to know the mechanism.

Dr. Wark, in reply to Dr. Blaubaum's question about nicotinic acid, said that he could offer no explanation, but that he had had a patient who had had the same experience with nicotinic acid, and who had finally required labyrinthectomy. Referring to recruitment, Dr. Wark said that he found the ordinary audiometer unsuitable for it. In reply to Dr. Cocks's question about stellate injection, Dr. Wark said that he had not heard of its being performed. In reply to Dr. Halliday's question about streptomycin, Dr. Wark said that he had had two patients in whom streptomycin had destroyed the labyrinthine function without affecting the cochlea; in one case streptomycin had been used therapeutically, and in the other the patient was suffering from cerebro-spinal fluid otorrhœa.

Dr. Schweiger again referred to recruitment; he said that it was one test which helped in the selection of patients for whom something definite could be done. In reply to Dr. Cossar's question, Dr. Schwieger said he thought that the ultimate prognosis was bound up with the lesion causing it, which was most likely to be central. Finally, in reply to the question about unconsciousness and vertigo, Dr. Schweiger said that he had seen it happen; moreover, Sir Charles Symonds was fond of the differential diagnosis in such cases. It was important that the vertigo persisted after the return of consciousness. The mechanism for that presumably was the explanation of what made the patient vomit. However, unconsciousness was infrequent in vertigo.

### Ligation for Severe Epistaxis.

DR. R. BLAUBAUM (Melbourne) read a paper entitled "Ligation for Severe Epistaxis" (see page 607).

Dr. von der Borch referred to a useful first aid measure for arteriosclerotic patients; control of the bleeding could be secured by a glove finger and catheter put in the nose and blown up with oxygen.

DR. J. H. SHAW (Melbourne) discussed the case of a patient who had had desperate nose bleeding, and on whom many procedures were performed; bleeding was finally controlled by infraction of the inferior turbinate and cauterization along the lateral nasal wall beneath it. That he thought had controlled the venous bleeding which was an important factor in many nasal hæmorrhages.

Dr. Cocks referred to telangiectasia. He said that he had employed œstrogenic hormone on two occasions, and no bleeding had occurred since.

Dr. Halliday further referred to three patients who were simultaneously in the Royal Prince Alfred Hospital, and who had had the treatment mentioned by Dr. Cocks, but with no success in any case.

DR. M. R. ROBERTSON (Lismore) said that on the contrary he had treated two patients successfully by the same method.

DR. F. F. ELLIS (Sydney) reported success with the use of hypotensive drugs.

Dr. Blaubaum, in reply, said he had used the glove-balloon method, but with no success. Hypotensive drugs, in his experience, had not always controlled the bleeding, even when the systolic blood pressure had been lowered to about 90 millimetres of mercury.

In reply to Dr. Shaw's suggestion about venous bleeding, Dr. Blaubaum said that he thought packing usually controlled it.

## Congresses.

### INTERNATIONAL SYMPOSIUM ON CARDIO-VASCULAR SURGERY.

AN International Symposium on Cardio-Vascular Surgery was held at Detroit, Michigan, United States of America, from March 17 to 19, 1955, under the auspices of Henry Ford Hospital. This third symposium sponsored by this hospital was held under ideal conditions in the fine new clinic building of 17 storeys recently completed. The auditorium held some five or six hundred persons, and many others had to be turned away. In addition to almost all the well-known names in the United States and Canada, European participants included Sir Russell Brock, Charles Rob, Charles Dubost, Clarence Crafoord and E. Sondergaard, and Australasian participants C. J. O. Brown and Douglas Robb. Mexico, Chile and Argentina also were represented. The local chairman was Conrad Lain, thoracic surgeon to Henry Ford Hospital, and the advisory committee was made up of Richard Bing, Stanley Gibson, Michael deBakey and Emile Holman. All aspects of the subject were discussed except machines for oxygenating extracorporeal blood and diseases of the venous system. Five subjects were dealt with in the form of panel discussions with a moderator and six to eight members in each.

A session on cardiac catheterization in the hands of Richard Bing revealed fundamental progress in the physiology and nutrition of heart muscle. Contributions on angiocardiography, presented by Swedish members, emphasized modern biplane methods with both cut films and rolls, and with both intravenous and selective injections to pick out special regions. Though costly, and to be watched on the score of radiation dosage, the fuller picture so obtained was remarkable.

The panel discussion on the treatment of pulmonary stenosis did not result in a decision in favour of Potts or Brock or Blalock, though large series of cases were reported. The latest variant was Henry Swan's series of open corrections under hypothermia. It was generally felt that with the prospect of safer conditions for open operations, present methods would be at least considerably altered.

In a symposium on adjustments between the systemic and pulmonary circulations in such conditions as early tetralogy of Fallot, and "atypical patent ductus", contributed to by Damann, Jesse Edwards, Helen Taussig and Ziegler, much information on physiological, pathological and clinical aspects was adduced and correlated.

The rather unpromising subject of transposition was reviewed by W. Mustard.

The panel discussion on interatrial septal defects was an exciting one, and participants included advocates of the various types of external suturing, with or without the guidance of a finger in the right atrium. Bailey's, Crafoord's and Sondergaard's modification of Sondergaard's operation, and Lain's double-pointed needle method were described, and also open operations under hypothermia for the difficult *"septum primum"* cases.

A particularly striking presentation from C. W. Lillehei, of Minneapolis, reported the closure of 22 ventricular septal defects (with seven deaths) under direct vision, the lungs of a donor being used by crossed circulation at normal temperatures. Among 36 donors so used there had been no fatality, only one fright. It was felt that the deepest secrets, or at least recesses, of the human heart had now been approached.

The morning of the second day was devoted to mitral valve disease, and included a panel discussion on late results of commissurotomy. Detailed physiological studies were reported differentiating mitral cases into those with purely mechanical obstruction, those with myocardial affection as well, and those with neither element seriously present, and a claim was made for the usefulness of electrokymography in recognizing regurgitation. Many useful points in the technique of operation for stenosis were brought out, but when it came to regurgitation it had to be admitted that nothing really practical was yet available, in spite of "much speaking". Bailey, Glover and Harken were the chief speakers on this subject. Hypothermia had been used only a little in mitral surgery.

Large series of cases of aortic stenosis treated surgically were reported, and the trend seemed to be in favour of entering the aorta by a cuff of some sort sutured to the incision in it, rather than by the transventricular route. The mortality, not unexpectedly, was high, but the cases were often desperate. Mortality was less in patients who had mitral stenosis relieved on the same occasion. Much detailed pathological anatomy was considered. Hufnagel reported 80 cases of insufficiency treated by the insertion of his plastic valve. In 50% of them the subjects were thoroughly "bad risks". There was a 20% hospital and 20% later mortality. The "oldest identity" was two and a half years post operation. The clicking noise was not a trouble to the patient, nor to his friends as long as he kept his mouth shut.

Hypothermia was debated by a panel of speakers, and physiological effects at various levels were established. The utility and difficulties of the various methods were considered, and the discussion issued in considerable support for blood-stream cooling after Delorme and Brock, though the various surface methods are in considerable vogue. The dangers of fibrillation and arrest and the chief indications for cooling were discussed.

The third day session was devoted to thoracic and abdominal aneurysms, and to peripheral vascular disease. Though a chill critical breeze passed through the remarks of Irving Page, of Cleveland, President-Elect of the American Heart Association, when he discussed the present state of our knowledge of the cause of atherosclerosis, the rest was a riot of buoyancy on the part of surgeons describing large series of resections and repairs and grafting. While homografts were accepted as effective for all sizes of arteries, there was general acceptance of artificial prostheses of "Orlon", "Dacron", "Nylon" and other fabrics as suitable, and possibly best, for aorta and iliac vessels. On the subject of peripheral occlusions, Rob, of London, gave a more cautious view of the indications, which he said were more favourable in the aorta and iliac and popliteal vessels than in the femoral vessels. DeBakey had operated successfully in four out of six cases of dissecting aneurysm in the chest. Methods of sterilization of homografts by $\gamma$ radiation and by "Beta-propiolactone" were advocated.

A dinner meeting, at which Willis Potts was the toastmaster and Sir Russell Brock the chief speaker, completed a memorable meeting. The proceedings are to be published in book form shortly by W. B. Saunders Company.

# Public Health.

## THE SECOND ASIAN MALARIA CONFERENCE OF THE WORLD HEALTH ORGANIZATION.

THE second Asian Malaria Conference for the Western Pacific and South-East Asia Regions, convened by the World Health Organization, was held at Baguio City, in the mountains of Northern Luzon, Philippine Islands, from November 15 to 24, 1954. The conference was concerned with technical matters, and thus differed from the first Asian Malaria Conference of the World Health Organization held at Bangkok in September, 1953, which dealt mainly with administration. It was originally proposed to hold the second conference in Taiwan (Formosa), but circumstances made it necessary to change the meeting place to the Philippines.

There were 42 participants at the conference. Some were invited by the World Health Organization, and others were sent by their governments, by the United Nations Children's Fund or by the United States Foreign Operations Administration. In addition members of the World Health Organization staff and a special consultant, Dr. P. F. Russell, attended the conference.

Dr. I. C. Fang, WHO Regional Director of the Western Pacific Region, opened the conference, and addresses were also given by Dr. W. M. Bonne, WHO Director of the Division of Communicable Disease Services, and by Dr. G. Sambasivan, on behalf of the Regional Director of the WHO South-East Asia Region. The Honourable Carlos F. Garcia, Vice-President of the Philippines, welcomed the participants to the Philippine Islands.

The conference elected Dr. Antonio Ejercito, Director of the Malaria Control Project, Department of Health of the Philippines, as its chairman, Dr. K. C. Liang, Director of the Taiwan Provincial Malaria Institute, Republic of China, as vice-chairman, and Dr. Soeparmo H.T., Director of the Malaria Institute of Indonesia, as rapporteur. Dr. F. J. Dy, Regional Malaria Adviser, WHO Western Pacific Regional Office, was elected as secretary. A drafting group was appointed to assist the rapporteur in preparing the report.

There was no formal presentation of papers at the conference, as documents had been submitted earlier and were available in roneoed form before the conference began. Each subject on the agenda was introduced by a selected participant, and discussion then followed. The language used was English, and microphones were readily available for every participant.

The first subject on the agenda dealt with vector problems of special interest. As the conference drew its participants from a wide area, a large number of species and subspecies of anophelines were discussed—namely, *A. minimus minimus, A. minimus flavirostris, A. maculatus, A. leucosphyrus leucosphyrus, A. leucosphyrus balabacensis, A. sundaicus* and the members of the *A. punctulatus* group (*A. punctulatus, A. farauti* and *A. koliensis*).

In China (Taiwan), Burma and Thailand malaria transmitted by *A. minimus minimus* has been successfully controlled by DDT residual spraying. In Thailand three years of spraying has resulted in almost complete elimination of this species in the greater part of the country. In the mountainous part of Vietnam it was necessary to augment residual spraying with the use of antimalarial drugs. Selective spraying of only parts of houses had been attempted in some areas, but the results are not yet definite.

In the Philippine Islands the vector, *A. minimus flavirostris*, was formerly considered not to rest indoors during the day to any extent. However, recent work by a WHO-assisted team on Mindoro has shown that treatment of houses with DDT will interrupt the transmission of malaria by this species.

In Malaya, where *A. maculatus* is the principal vector, a field trial with DDT or BHC used as a residual insecticide reduced but did not stop malaria transmission. In Sarawak, where *A. leucosphyrus leucosphyrus* is the main vector, preliminary work has shown that residual spraying may control malaria, but further observations are required. Selective clearing of jungle by herbicides and cultivation was considered to be worthy of trial in north Borneo, where *A. leucosphyrus balabacensis* is the chief vector.

In some parts of Indonesia the main vector, *A. sundaicus*, has disappeared after the use of DDT as a residual spray, but in two areas there were reports of the

development of resistance to DDT by this species. In both these areas DDT had previously been used as a larvicide. This is the first report of the development of resistance to DDT by anophelines in the South-East Asia and Western Pacific Regions. A behaviouristic change on the part of *A. sundaicus* was reported from South Java, where this species now avoids prolonged contact with DDT-treated surfaces.

The *A. punctulatus* group contains the chief vectors of malaria in the South-West Pacific Area—New Guinea, Solomon Islands and New Hebrides. Recent studies on the behaviour of members of this group and the results of a pilot project with DDT used as a residual spray in Netherlands New Guinea give hopes that this method may be successful in controlling malaria in New Guinea. In the New Hebrides, where the vector is *A. farauti*, satisfactory control has already been obtained in some areas where DDT residual spraying has been used.

The development of anopheline resistance to DDT was carefully studied by the conference, which endorsed the conclusion of the WHO Symposium at Rome in 1953 on "Control of Insect Vectors of Disease" that "the use of chemically related insecticides against both the adults and the larvæ should not be carried out simultaneously in the same area, except in cases of emergency".

There were many problems upon which research was necessary. Some work was reported on the behaviour of some of the vector species. Suspicion was voiced on the possible occurrence of subspecies or races with different biting and resting habits—these races not being morphologically distinguishable. The opinion was expressed that the problems of colonization of vector species must be investigated for the carrying out of biological studies in cross-breeding, resistance to insecticides and adult behaviour. It was considered that the use of trap huts was a valuable method of study of anopheline behaviour and reaction to residual insecticides.

Observations are required to determine whether the relapsing vivax malaria of New Guinea is localized to that island or is widespread throughout the two regions. Little is known about the response of malaria parasites in the different countries to proguanil and pyrimethamine, drugs to which malaria parasites may become resistant. Further study is required, if the opportunity presents itself, on the morphology of the malaria parasites throughout the area to determine if there are geographical varieties.

Studies on the therapy of relapsing vivax malaria are desirable to determine the response of this species from different areas in the regions. Similarly, further therapeutic trials with chloroquine and amodiaquine in falciparum infections are necessary.

Research in control methods should be directed at lowering the cost of control and improving the method of application of residual insecticides. There is, too, a place for limited studies in naturalistic methods of malaria control.

It was brought to the notice of the conference that, in parts of New Guinea, transmission rates are very high with a high spleen rate in both children and adults. This finding is at variance with the classification of the degrees of malarial endemicity defined by the WHO Malaria Conference for Equatorial Africa. The conference suggested that this problem be further studied by observations on transmission rate and the development of immunity.

Drugs give a high degree of protection against malaria in certain groups, but take second place to residual spraying in the control of rural malaria. However, drugs have a place in residual spraying programmes, as, for example, in the eradication of residual reservoirs of infection when transmission has been interrupted, and in some areas where the effect of residual spraying is slow. The use of drugs is the best method of dealing with a malaria epidemic; in addition, spraying with residual insecticides should begin at once. Seasonal migration of populations from controlled to uncontrolled areas is also an indication for the use of suppressive drugs. Some countries still hold large stocks of quinine or mepacrine, which are less effective than chloroquine and amodiaquine, but, if used efficiently, can usefully supplement residual spraying programmes.

Reports were received from 23 countries on their malaria control programmes. These reports covered a total population of 659,000,000 people, and of these 276,000,000 live in malarious areas. In 1953 a total of 80,000,000 (29% of those at risk) had been protected against malaria. In the South-West Pacific Area the percentage protected of those at risk was about 2. In Ceylon there is a population of over 8,000,000 people, of whom 3,000,000 live in malarious areas. A programme for malaria control with residual spraying of DDT was begun in 1946. Malaria has now been almost eradicated from this country, and in a large area spraying has been discontinued and replaced by careful surveillance. The cost of this programme was 2 cents (United States) *per capita* per year. In the various countries where the national malaria control programmes use residual spraying the cost *per capita* per year for persons protected has usually ranged between about 10 and 20 cents (United States).

The economic burden of malaria upon individuals, communities and nations was considered by the conference to be costly because of its effects on the physical, economic and social health. There is need, however, for more definite methods of measuring the cost of malaria. Successful malaria control is credited with reduction in infantile mortality and general death rates, reduced absenteeism, reduced incidence in other insect-borne diseases, increased use of land, increase in tourist traffic and increased cooperation in public health programmes. The effect of malaria control on world population and food production was considered by the conference, and it was agreed that no one could predict the total population that the world could carry if all potential supplies were properly used. No one was in a position to be able to determine which areas should or should not be subject to malaria control. Malaria control must be integrated with other public activities designed to bring about improvement in community welfare. The conference considered malaria control a sound economic investment. "No country with a serious malaria problem can afford *not* to control malaria."

National malaria control programmes require adequate trained personnel, and some of these may be trained in other countries. However, certain groups of workers should be trained locally and training should thus be part of the control programme. Besides technical personnel there is need for administrators, who may be more efficient if given some training in malaria control.

The objective of a national malaria campaign is eradication of the disease; and if this is to be attained, coordinated effort is required. Now that it is realized that resistance to DDT may be developed by anophelines, campaigns must be planned as efficiently as possible and with the aim of discontinuing residual spraying as soon as possible. All areas should be brought under control simultaneously. There must be adequate personnel, equipment, transport and supplies at all operational centres with smoothly working channels of communication and responsibility. This necessitates a national malaria service with full authority and technical responsibility. Coordination is necessary with the military and air forces. Between countries also coordination is necessary. Nation-wide malaria programmes require the support of adequate legislation; antimalarial enactments already in force may need revision to bring them into line with modern methods. Unless especially constituted, malaria control programmes should not be encumbered with the additional responsibility of controlling other insect-borne diseases.

The conference agreed upon the necessity for standardization, especially in reporting. Attention was drawn to the WHO monograph "Malaria Terminology" and the proposals of the WHO Malaria Conference in Equatorial Africa, 1950.

It was agreed that residual spraying should be interrupted as soon as possible; the criteria which determined when this should be done were a matter for decision by competent local authorities will versed in the local epidemiology of malaria. When spraying has been stopped there must be adequate and competent surveillance for a number of years afterwards, in addition to adequate staff, supplies, transport *et cetera* for the immediate resumption of spraying if the need arises. In some areas it may not be possible to eradicate malaria by residual spraying, and the conference suggested that it might be advisable in such areas to abandon the use of residual spraying to avoid development of resistance to the insecticide. Other methods of control should be substituted. It was emphasized that when resistance to DDT had developed, replacement by dieldrin, chlordane, BHC or lindane might not be effective for long. Experience with the chlorinated hydrocarbons suggests that when resistance has developed towards one, it may rapidly extend to the others. It is inadvisable to continue indefinitely to attempt malaria control by residual spraying with chlorinated hydrocarbon insecticides.

As a result of these discussions various recommendations and conclusions were drawn up by the conference. Those which particularly affect the South-West Pacific Area will

be recorded here. It was recommended that experimental work on the control of malaria transmission by the *A. punctulatus* group by residual insecticides be continued and extended to other areas. The conference also recommended that, in the absence of an emergency, chlorinated hydrocarbons be not used either concurrently or consecutively as both adulticides and larvicides in a given area. It was concluded that more information was needed on the sensitivity of *Plasmodium vivax* in the two regions to primaquine and pyrimethamine. The conference further recommended (i) that national malaria services be set up with appropriate authority and full technical responsibility in the field of malaria control and (ii) that national malaria advisory committees be established wherever necessary to coordinate all plans and activities of malaria control within the country. The final recommendation of the conference was that the ultimate goal of a nation-wide malaria control programme be the eradication of the disease.

When the conference had closed, a visit was made by the participants to the Malaria Field Centre at Tala, near Manila, where training courses are carried on for technical personnel. In addition, a party proceeded by air to Taiwan (Formosa) to observe the work being done by the national malaria control service on that island.

Participants at the conference from the South-West Pacific Area comprised Dr. H. de Rook, Malariologist of the Netherlands New Guinea Petroleum Company, Sorong, Netherlands New Guinea, Dr. D. Metselaar, Senior Government Malariologist, Netherlands New Guinea, and Dr. Robert H. Black, of the School of Public Health and Tropical Medicine, Sydney. Amongst the observers was Professor L. E. Rozeboom, who had experience with wartime malaria in the Solomon Islands.

## DANGEROUS DRUGS: PARAGRAPH (2) OF THE SIXTH SCHEDULE TO THE POISONS ACT, 1928, OF VICTORIA.

THE following proclamation has been published in the *Victoria Government Gazette*, Number 93, of March 23, 1955.

By virtue of the powers conferred by section thirty-eight of the *Poisons Act* 1928 (No. 3748), as amended by section five of Act No. 3918, I, the Governor of the State of Victoria in the Commonwealth of Australia, by and with the advice of the Executive Council of the said State and on the recommendation of the Pharmacy Board of Victoria, do by this my Proclamation add the names of the following substances and preparations to paragraph (2) of the Sixth Schedule of the *Poisons Act* 1928 as amended by any Act or Enactment, namely:

1. Fluorides of metals (including ammonium fluoride) intended for ingestion; their solutions, preparations and admixtures.
2. Folic Acid Antagonists such as Teropterin, Aminopterin, Amethopterin, Orthopterin; the solutions, preparations and admixtures of these antagonists or of any of their derivatives by whatever names such folic antagonists are described.
3. Cytotoxic substances with blood destroying and/or anti-cancer properties such as Mustine Hydrochloride, Lekamin, Trillekamin, Triethylene Melamine, T.E.M., Tepa, Thiotepa, G49, Triethylene Phosporamide, 6-mercaptopurine, Purinethol, Thioguanine, Myleran; the solutions, preparations and admixtures of these substances and/or any of their derivatives by whatever name such cytotoxic substances are described and for whatever purposes they are intended.
4. Chlorpromazine and its salts; their solutions, preparations and admixtures,

and declare that Division 2 of Part III. of the *Poisons Act* 1928 as amended by Act No. 3918 shall apply to the substances and preparations so added in the same manner as such Division applies to the substances and preparations included in the said paragraph (2).

Given under my Hand and the Seal of the State of Victoria aforesaid, at Melbourne, this sixteenth day of March, in the year of our Lord one thousand nine hundred and fifty-five, and in the fourth year of the reign of Her Majesty Queen Elizabeth II.

(L.S.) DALLAS BROOKS.

By His Excellency's Command,

WM. BARRY,
Minister of Health.

# British Medical Association News.

## THE BRITISH MEDICAL ASSOCIATION ANNUAL GENERAL MEETING, 1935, PRIZE.

THE British Medical Association Annual General Meeting, 1935, Prize is open to all members of the Association in Australia and its Mandated Territories and in New Zealand, and to any graduate of an Australian university who is a member of the Association.

The prize is to be awarded for that contribution towards the study of problems in the medical and allied sciences adjudged to be of greatest merit. If no contribution is considered to be of sufficient merit, the prize may not be awarded.

The prize shall consist of a medal suitably inscribed, together with the balance of the income from the capital fund available for distribution at the time.

The period during which any contribution for consideration may be made shall be the three years ending on December 31 immediately preceding a meeting of the Australasian Medical Congress (British Medical Association).

This is a triennial prize, and it is intended that it may be awarded for the second time at the meeting of Congress to be held in Sydney in August, 1955.

To minimize the risk that a contribution of high value may be overlooked, it will be appreciated if the contributor of any such work complying with the above conditions be nominated to the Prize Committee. It is requested that such nominations be forwarded not later than April 30, 1955, to Dr. J. P. Major, Chairman of the Prize Committee, British Medical Association (Victorian Branch), 426 Albert Street, East Melbourne, C.2.

## MEDICAL PRACTITIONERS' FLOOD RELIEF FUND.

THE following is a list of contributors to the Medical Practitioners' Flood Relief Fund opened by the Council of the New South Wales Branch of the British Medical Association to assist medical practitioners who suffered loss in the recent disastrous floods in New South Wales.

| | £ | s. | d. | | £ | s. | d. |
|---|---|---|---|---|---|---|---|
| Byrne, Ethel .. | 10 | 10 | 0 | Nelson, T. Y. .. | 10 | 10 | 0 |
| Macdonald, R. H. | 10 | 10 | 0 | Randall, C. .. .. | 2 | 2 | 0 |
| Syred, R. H. .. | 5 | 5 | 0 | Hirsz, J. .. .. .. | 2 | 2 | 0 |
| Freidman, E. H. | 2 | 0 | 0 | Schofield, J. A. V. | 2 | 2 | 0 |
| O'Loan, John .. | 1 | 1 | 0 | Merrington, H. N. | 25 | 0 | 0 |
| Downes, G. B., | | | | Allison, J. R. .. | 5 | 5 | 0 |
| Arnheim, R. F. | | | | Bye, W. A. .. .. | 5 | 5 | 0 |
| G., and Donnan, | | | | Haneman, B. .. | 10 | 10 | 0 |
| B. W. .. .. .. | 10 | 10 | 0 | Jennaway, R. .. | 5 | 5 | 0 |
| Browne, G. .. .. | 2 | 2 | 0 | Quirk, D. .. .. | 1 | 1 | 0 |
| Symons, C. Y. .. | 2 | 2 | 0 | Poate, Sir Hugh .. | 31 | 10 | 0 |
| Wallman, N. S., | | | | Hodge, A. H. .. | 5 | 5 | 0 |
| and Becker, T. | 5 | 5 | 0 | Turek, M. .. .. | 1 | 1 | 0 |
| Glennie Holmes, R. | | | | Pockley, Eric .. | 5 | 5 | 0 |
| M. .. .. .. | 5 | 5 | 0 | Sacks, M. L. .. .. | 1 | 1 | 0 |
| Simmons, W. F. .. | 25 | 0 | 0 | Smith, G. K. .. .. | 3 | 3 | 0 |
| Grieve, H. R. R. | 10 | 10 | 0 | Gilchrist, Marjorie | 2 | 2 | 0 |
| Glennie Holmes, | | | | Jefferis, R. E. .. | 5 | 5 | 0 |
| T. A. .. .. .. | 20 | 0 | 0 | Goulston, S. J. M. | 2 | 2 | 0 |
| Dowling, J. M. .. | 10 | 0 | 0 | Thomson, R. M. .. | 1 | 1 | 0 |
| Brenner, M. .. .. | 10 | 10 | 0 | Brass, S. .. .. | 2 | 2 | 0 |
| Charlton, H. W. .. | 2 | 2 | 0 | Caplan, D. .. .. | 3 | 3 | 0 |
| Brookes, W. L. .. | 5 | 0 | 0 | Benjamin, N. F. .. | 5 | 5 | 0 |
| Smith, Kenneth .. | 2 | 0 | 0 | Morris, G. .. .. | 5 | 5 | 0 |
| Walker, J. .. .. | 2 | 2 | 0 | Allison, R. W. .. | 1 | 1 | 0 |
| Howell, F. J., .. | 5 | 0 | 0 | Traill, A. J. .. .. | 3 | 3 | 0 |
| Edye, B. T. .. .. | 26 | 5 | 0 | Boulton, N. P. .. | 3 | 3 | 0 |
| Lawes, F. A. E. | 5 | 5 | 0 | Dowe, J. B. .. .. | 5 | 5 | 0 |
| Edwards, M. L. .. | 5 | 5 | 0 | Solomon, I. .. .. | 5 | 5 | 0 |
| Abramovich, H. .. | 20 | 0 | 0 | Lovell, S. H. .. | 5 | 5 | 0 |
| Williams, D. A. .. | 5 | 5 | 0 | Hobson, G. E. .. | 5 | 5 | 0 |
| Hay, V. .. .. .. | 1 | 1 | 0 | Ingram, B. R. .. | 3 | 3 | 0 |
| Cole, C. .. .. .. | 5 | 0 | 0 | Davidson, R. M. .. | 3 | 3 | 0 |
| Corbett, W. H. .. | 1 | 0 | 0 | Maitland, D. G., | | | |
| Johnston, M. Grace | 5 | 5 | 0 | and Booth, E. A. | 2 | 2 | 0 |
| Chambers, R. G. .. | 3 | 3 | 0 | Foley, H. J. .. .. | 10 | 0 | 0 |
| Hock, W. J. .. .. | 5 | 5 | 0 | Kline, Z. .. .. .. | 1 | 1 | 0 |
| May, Leonard .. | 10 | 10 | 0 | Varvaressos, D .. | 2 | 2 | 0 |
| Genzel, J. .. .. | 1 | 1 | 0 | Breinl, W. .. .. | 5 | 5 | 0 |
| Saxby, W. H. .. | 2 | 2 | 0 | Woolnough, A. R. | 5 | 5 | 0 |

| | £ | s. | d. |
|---|---|---|---|
| Hudson, C. B. | 3 | 3 | 0 |
| Scougall, S. H., and Colvin, G. S. | 5 | 5 | 0 |
| Harpur, M. D. H. | 5 | 5 | 0 |
| Warnock, C. M., and Brass, R. | 1 | 1 | 0 |
| Chapman, Patricia | | 10 | 6 |
| Parkinson, C. K. | 2 | 2 | 0 |
| Willis, H. H. | 2 | 2 | 0 |
| Smith, Winston | 10 | 0 | 0 |
| Hipsley, P. D. | 5 | 5 | 0 |
| Murray-Will, E. | 26 | 5 | 0 |
| Murphy, K. M. | 5 | 5 | 0 |
| Shortridge, D. | 5 | 5 | 0 |
| Winton, R. R. | 5 | 0 | 0 |
| MacDonald, W. M. C. | 5 | 5 | 0 |
| Stuckey, E. S. | 5 | 5 | 0 |
| Bryant, A. L. | 5 | 0 | 0 |
| White, H. J. | 5 | 5 | 0 |
| Miller, D. | 10 | 0 | 0 |
| Boydell, W. H. | 2 | 2 | 0 |
| Selby, George | 3 | 3 | 0 |
| Gibson, E. W. | 2 | 2 | 0 |
| Grace, S. St. J. | 2 | 2 | 0 |
| Roberts, A. P. | 5 | 5 | 0 |
| Thorburn, C. H. | 2 | 2 | 0 |
| Irwin, R. S. | 1 | 1 | 0 |
| Solomon, H. J. | 3 | 0 | 0 |
| Harrison, C. W. | 2 | 2 | 0 |
| Beazley, R. N. | 4 | 4 | 0 |
| Fox, W. R. F. | 5 | 5 | 0 |
| Walker, J. | 3 | 3 | 0 |
| Mason, J. H. | 3 | 3 | 0 |
| MacCallum, W. P. | 5 | 5 | 0 |
| Hoets, J. | 5 | 5 | 0 |
| Archibold, G. | 2 | 2 | 0 |
| Cambourn, P., Irvine, A. F., and Binks, R. | 5 | 5 | 0 |
| Geikie, G. C. | 1 | 1 | 0 |
| Barry, K. L. | 2 | 2 | 0 |
| Pfeiffer, G. H. | 10 | 10 | 0 |
| Henniker, R. N. | 2 | 2 | 0 |
| Morrow, A. W. | 10 | 0 | 0 |
| Pittar, R. | 5 | 5 | 0 |
| Johnson, M. H. | 5 | 5 | 0 |
| Cox, C. B., and Rudd, G. V. | 25 | 0 | 0 |
| Heydon, G. A. M. | 10 | 0 | 0 |
| Armstrong, K. B. | 5 | 5 | 0 |
| Ferguson, D. A. | 2 | 2 | 0 |
| Steigrad, J. | 5 | 5 | 0 |
| Gill, R. C. | 5 | 5 | 0 |
| Mutton, M. V. | 1 | 1 | 0 |
| Frecker, B. E. | 5 | 5 | 0 |
| Wiseman, J. E. | 3 | 3 | 0 |
| Tinckam, M. N. | 10 | 10 | 0 |
| Mackay, R. G. | 2 | 2 | 0 |
| Middleton, A. W. | 3 | 3 | 0 |
| Bray, Sylvia | 3 | 3 | 0 |
| Armstrong, C. J. B. | 5 | 5 | 0 |
| Belisario, J. C. | 5 | 5 | 0 |
| Edye, Beryl | 1 | 0 | 0 |
| Edwards, L. L. | 10 | 0 | 0 |
| Stening, Warwick | 5 | 5 | 0 |
| Barr, K. A., Levene, M. M., and Guyot, J. R. B. | 10 | 10 | 0 |
| Zeigler, Anna | 2 | 2 | 0 |
| Droulers, J. P. | 3 | 3 | 0 |
| Bulteau, V. | 5 | 5 | 0 |
| Lush, P. | 1 | 0 | 0 |
| Ridley, Brenda | 5 | 5 | 0 |
| Allen, M. | 10 | 10 | 0 |
| Balzer, J. and N., and Frost, T. J. | 5 | 5 | 0 |
| Barry, H. C. | 2 | 2 | 0 |
| Campbell, C. A. K. | 10 | 10 | 0 |
| Gunther, C. E. M. | 5 | 5 | 0 |
| Harrison, G. | 2 | 0 | 0 |
| Henchman, D. C. | 1 | 1 | 0 |
| Hornbrook, A. F. | 1 | 1 | 0 |
| Humphery, E. M. | 5 | 0 | 0 |
| Hunter, R. J. | 2 | 2 | 0 |
| Landor, J. V. | 1 | 1 | 0 |
| Latham, O. | 5 | 5 | 0 |
| McGregor, R. | 3 | 3 | 0 |
| Pittar, Doreen, and Leckie, B. D. | 2 | 2 | 0 |
| MacCulloch, H. T. C. | 5 | 5 | 0 |
| McLaren, N. E. | 5 | 5 | 0 |
| Martin, C. J. | 5 | 5 | 0 |
| O'Brien, J., and Dynon, G. P. | 5 | 0 | 0 |
| Palmer, E. C. | 2 | 2 | 0 |
| Pirie, J. M. G. | 5 | 5 | 0 |
| Rainsford, F. G. | 2 | 2 | 0 |
| Reiner, Z. | 3 | 0 | 0 |
| Rich, B. M. | 1 | 1 | 0 |
| Robertson, S. E. J. | 5 | 5 | 0 |
| Rosenfield, F. | 3 | 3 | 0 |
| Trousdale, D. H. | 5 | 5 | 0 |
| Walker, C. | 5 | 5 | 0 |
| Walker, R. L. | 2 | 2 | 0 |
| Maclean-Lilley, L. A. | 2 | 2 | 0 |
| Feher, S. | 5 | 5 | 0 |
| Lachlan, W. | 2 | 2 | 0 |
| Bassett, R. | 3 | 3 | 0 |
| Epps, W. G. H. | 5 | 5 | 0 |
| Weisz, A. | 2 | 2 | 0 |
| Jones, B. L. | 5 | 0 | 0 |
| Atkins, W. T. G. | 2 | 2 | 0 |
| Blomfield, E. C. | 5 | 0 | 0 |
| Archdall, M. | 5 | 5 | 0 |
| Kater, Sir Norman | 2 | 2 | 0 |
| Chambers, R. | 5 | 0 | 0 |
| Hoy, R. J. | 5 | 0 | 0 |
| Joseph, N. | 3 | 3 | 0 |
| Opitz, F. P. | 5 | 5 | 0 |
| Peate, D. L. | 10 | 10 | 0 |
| Wyndham, N. | 5 | 5 | 0 |
| Burns, F. A. | 5 | 5 | 0 |
| Sinclair, R. W. | 2 | 2 | 0 |
| Burfitt, B. J. | 5 | 5 | 0 |
| Moon, A. A. | 5 | 5 | 0 |
| Spragg, G. S. | 2 | 2 | 0 |
| Rundle, F. F. | 5 | 5 | 0 |
| Bowen-Thomas, G. | 5 | 0 | 0 |
| Rychter, O. | 3 | 3 | 0 |
| Collins, Sir Archibald | 2 | 2 | 0 |
| McKay, R. M. | 3 | 3 | 0 |
| Read, W. H. | 5 | 0 | 0 |
| Wilkins, T. D. | 5 | 0 | 0 |
| Mahon, A. L. | 5 | 5 | 0 |
| Joseph, L. | 5 | 5 | 0 |
| Wherrett, S. W. | 10 | 10 | 0 |
| Crawford, C. G. | 5 | 5 | 0 |
| Brown, D. S. | 3 | 3 | 0 |
| Anonymous | 5 | 0 | 0 |
| Lundie, A. J. | 5 | 5 | 0 |
| King, K. J. | 2 | 2 | 0 |
| Freeborn, W. | 3 | 3 | 0 |
| Roger, J. R. O. | 3 | 3 | 0 |
| Harris, H. R., Holland, E. P., Mulhearn, N. St. C., Robinson, K. V., Walters, D. J., and Rae, J. L. | 10 | 10 | 0 |
| Burne, A. R. K. | 5 | 0 | 0 |
| Doyle, P. H. | 2 | 2 | 0 |
| Rush, S. J. | 3 | 3 | 0 |
| Dash, E. G. | 5 | 5 | 0 |
| Rutherford, Dorothy | 3 | 3 | 0 |
| Harrison, K. S. | 5 | 5 | 0 |
| Jobson, P. L. | 2 | 2 | 0 |
| Gibb, H. I. | 2 | 0 | 0 |
| Alexander, M. S. | 5 | 5 | 0 |
| Paterson, D. B. | 1 | 1 | 0 |
| Alexander, K. W. | 10 | 10 | 0 |
| Gregg, A. E. | 3 | 3 | 0 |
| Buchanan, W. F. | 10 | 0 | 0 |
| Fraser, W. H. | 10 | 10 | 0 |
| Beveridge, Ruby S. | 10 | 0 | 0 |
| Parker, K. S., Dun, C. W. S., Jones, K. S., Williams, D. C., Higham, R. H., Gosby, S. M., and Bennet, V. J. | 10 | 10 | 0 |
| Blashki E. P. | 5 | 0 | 0 |
| Holt, G. C. | 10 | 0 | 0 |
| Clark, E. D. | 5 | 5 | 0 |
| Tunley, L. W. and M. E. | 5 | 5 | 0 |
| Isles, J. L. | 1 | 1 | 0 |
| Meers, H. N. | 1 | 1 | 0 |
| Barrett, C. G. | 5 | 0 | 0 |
| Coolican, R. E. | 10 | 10 | 0 |
| Page, I., and Truscott, M. | 10 | 10 | 0 |
| Corin-Shedden, K. Ruth | 2 | 2 | 0 |
| Cowdery, R. D. | 2 | 2 | 0 |
| Amphlett, Julia | 5 | 5 | 0 |
| Barbour, J. | 7 | 7 | 0 |
| Parr, L. J. A. | 2 | 2 | 0 |
| McKay, Frances C. B. | 2 | 2 | 0 |
| Lyle, Jean | 2 | 2 | 0 |
| Peterson, B. H. | 1 | 1 | 0 |
| McManis, A. G. | 2 | 2 | 0 |
| Sara, C. A. | 5 | 5 | 0 |
| Rawle, K. C. T. | 10 | 10 | 0 |
| Mollenhauer, P. L. | 1 | 1 | 0 |
| Whitfeld, S. G. | 5 | 0 | 0 |
| Sheehan, A. H. | 5 | 5 | 0 |
| Sullivan, J. F. | 5 | 5 | 0 |
| Morris, J. | 2 | 0 | 0 |
| Outhred, K. G. | 4 | 4 | 0 |
| Hotten, W. I. T. | 5 | 5 | 0 |
| Cull, F. M. | 10 | 0 | 0 |
| Macintosh, A. H. | 5 | 0 | 0 |
| Bull, I. L. | 3 | 3 | 0 |
| Harris, J. | 10 | 0 | 0 |
| Kingsley, John | 5 | 5 | 0 |
| Ryan, F. P. | 10 | 10 | 0 |
| Abbott, L. G. | 1 | 1 | 0 |
| ten Seldam, R. E. J. | 1 | 1 | 0 |
| McLaughlin, W. P., and Murray-Prior, H. B. | 10 | 10 | 0 |
| Howell, F. J., Smith, G. S., and White, W. B. | 5 | 5 | 0 |
| Manion, N. | 3 | 3 | 0 |
| Priestley, J. H. | 5 | 5 | 0 |
| Dodson, L. F. | 1 | 1 | 0 |
| Aitkens, G. N. M. | 10 | 10 | 0 |
| Anderson, Edith | 2 | 2 | 0 |
| Willcocks, G. C. | 10 | 0 | 0 |
| Thorp, J. H. | 5 | 5 | 0 |
| Sharp, Alan | 2 | 2 | 0 |
| Kaye, A. M. | 2 | 2 | 0 |
| Johnson, Adrian | 10 | 10 | 0 |
| Matthews, H. D. | 10 | 10 | 0 |
| Anderson, Phyllis M. | 2 | 2 | 0 |
| Grossy, M. | 5 | 0 | 0 |
| Lawrie, L. | 5 | 5 | 0 |
| Spencer, S. L. | 10 | 10 | 0 |
| Waddy, Nanette | 5 | 5 | 0 |
| Cuthbert, H. W. | 5 | 5 | 0 |
| Friedman, I. M. | 5 | 0 | 0 |
| Buchanan, Lyle | 2 | 2 | 0 |
| Gillespie, A. D. | 2 | 2 | 0 |
| Hurman, Edith M. | 3 | 3 | 0 |
| Rivett, E. W., R. A. and H. M. | 15 | 15 | 0 |
| Goldschlag, F. | 2 | 2 | 0 |
| Geeves, R. B. | 5 | 5 | 0 |
| Allworth, C. T. | 3 | 3 | 0 |
| Gregg, Sir Norman | 5 | 5 | 0 |
| De Garis, Mary C. (Victoria) | 5 | 5 | 0 |
| Shanasy, Frank (Victoria) | 10 | 10 | 0 |
| Hugh-Smith, W. A. | 5 | 0 | 0 |
| Perendi, S. | 5 | 0 | 0 |
| Scarlett, G. A. | 5 | 5 | 0 |
| Ashby, G. W., Latimer, Thelma L., and Pearson, B. C. | 10 | 10 | 0 |
| Mitchell, B. Aileen | 10 | 10 | 0 |
| Greenaway, T. M. | 10 | 10 | 0 |
| Burke, Nora (Western Australia) | 2 | 2 | 0 |
| Grieve, D. | 1 | 1 | 0 |
| Schnieder, O. H., Richards, C. R. B., Thorburn, C. H., and de Meyrick, G. W. | 21 | 0 | 0 |
| Ryan, H. A. | 5 | 5 | 0 |
| McQueen, E. N. | 10 | 0 | 0 |
| Granger, Marjorie B. | 2 | 2 | 0 |
| Dent, J. A. S. | 10 | 10 | 0 |
| Gollan, L. N. (Tasmania) | 5 | 5 | 0 |
| Crakanthorp, J. S. | 5 | 5 | 0 |
| Phillips, J. B. | 2 | 2 | 0 |
| Bligh, R. G. | 2 | 2 | 0 |
| Gray, J. B. | 5 | 5 | 0 |
| Champion, C. G., and Smith, A. K., Chancellor, A. H., and Paver, K. | 3 | 3 | 0 |
| Iceton, S. G. | 5 | 5 | 0 |
| Wilshire, J. M. | 2 | 2 | 0 |
| Anonymous | | 10 | 0 |
| Swain, I. H. F. | 5 | 0 | 0 |
| Mitchell, D. H. | 5 | 5 | 0 |
| Hewitt, T. G., and Lawrance, K. G. | 21 | 0 | 0 |
| Bathgate, D. D. | 10 | 10 | 0 |
| Murray, A. J. | 20 | 0 | 0 |
| Holcombe, T. E. Y. | 3 | 3 | 0 |
| Helms, Karen | 2 | 2 | 0 |
| Blackburn, Sir Charles | 10 | 10 | 0 |
| Lance, A. L. | 2 | 2 | 0 |
| Woolnough, J. | 2 | 2 | 0 |
| Uther, F. B. | 5 | 5 | 0 |
| O'Reilly, C. F. | 1 | 0 | 0 |
| Voss, K. B. | 5 | 5 | 0 |
| Fitzpatrick, E. B. | 5 | 5 | 0 |
| Tandy, Dorothy R. | 2 | 2 | 0 |
| Stephens, F. G. N. | 15 | 0 | 0 |
| Furner, C. R. | 10 | 10 | 0 |
| Daniel, P. L. | 5 | 5 | 0 |
| Elliott, Mervyn | 2 | 2 | 0 |
| Robertson, M. | 1 | 1 | 0 |
| Kelly, Michael (Victoria) | 21 | 0 | 0 |
| Muscio, A. | 3 | 0 | 0 |
| Pittar, D. J. | 2 | 2 | 0 |
| Chesterman, J. N. | 2 | 2 | 0 |
| Trenerry, E. | 10 | 10 | 0 |
| Beale, J. G. Morris (Queensland) | 5 | 5 | 0 |
| Sear, H. | 10 | 0 | 0 |
| McKellar Hall, R. D. (Western Australia) | 20 | 0 | 0 |
| British Medical Association, London, £500 sterling | 625 | 0 | 0 |
| Deakin, J. E. F. | 5 | 5 | 0 |
| Pearson, H. H. | 5 | 5 | 0 |
| Smith, N. D. J., and Potts, L. G. | 2 | 2 | 0 |
| Rawle, R. M. | 5 | 5 | 0 |
| Whiting, T. K. S. | 3 | 0 | 0 |
| Macintosh, A. M. | 2 | 2 | 0 |
| Brown-Craig, F. | 3 | 3 | 0 |
| Cameron, Ian | 3 | 3 | 0 |
| McDermott, C. A. | 5 | 5 | 0 |
| Cooley, Beryl G. | 2 | 0 | 0 |
| Vanderfield, G. K. | 3 | 3 | 0 |
| Vickery, C. E. | 2 | 2 | 0 |
| Davis, Clyde | 5 | 5 | 0 |
| Niesche, F. W. | 5 | 5 | 0 |
| Sharp, Henry | 5 | 5 | 0 |
| George, S. | 5 | 5 | 0 |
| Stokes, E. H. | 5 | 5 | 0 |
| Chenhall, F. N. | 5 | 5 | 0 |
| Graham, E. D. | 1 | 1 | 0 |
| Barber, E. | 1 | 0 | 0 |
| Skeoch, Hugh, F.R.C.S. (Lond.) | 100 | 0 | 0 |
| Hunter, J. G. | 5 | 5 | 0 |
| Uren, H. K. | 10 | 10 | 0 |
| Geeves, R. C. | 2 | 2 | 0 |
| MacNaught, I. W. | 5 | 5 | 0 |

| | £ s. d. | | £ s. d. |
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| Rae, R. K. .. .. | 5 5 0 | Hughes, R. T. C. | 2 2 0 |
| Lawson, D. W. .. | 15 15 0 | Phillips, B. J. .. | 5 0 0 |
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## SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Royal Children's Hospital, Carlton, on November 10, 1954. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital.

### Diabetic Clinic.

DR. GERALDINE AMIES gave a demonstration on the use of the newer depot insulins—NPH and the three of the *lente* series—in juvenile diabetes. Their time activity was compared with that of the older types of insulin, and the indications and contraindications for use of the newer preparations were discussed. Blood sugar serial determinations were shown of a group of labile diabetic children who had been treated with them for eighteen months.

### Dermatological Demonstration.

DR. ARTHUR J. DAY showed "Kodachrome" slides demonstrating the treatment of various types of hæmangiomata with pictures of patients before and after treatment by X-radiation. He stressed the importance of early treatment, when often simple treatment by carbon dioxide snow was effective. He stated that after two years of age, surgery was the treatment of election.

The differential diagnosis and treatment of infantile eczema, *urticaria papulosa, impetigo contagiosa, granuloma annulare* and *molluscum contagiosum* were also demonstrated.

DR. F. BAUER showed patients to demonstrate the differential diagnosis of patchy loss of hair.

### Hypothyroidism and Colloid Goitre.

DR. MONA BLANCH presented five children who illustrated various difficulties in the diagnosis and treatment of hypothyroidism. The differential diagnosis between cretinism, mongolism, gargoylism and the backward child was stressed; also the necessity for adequate and continuous medication with a standard preparation of dry thyroid.

Dr. Blanch's second group of patients consisted of three children with enlarged thyroid glands. The first was a baby of eleven months, who had been born with a colloid goitre so large that delivery had been difficult. His mother had been treated for hyperthyroidism throughout pregnancy with 0·1 grain of thiouracil on alternate days. Despite administration of *Thyroideum Siccum*, one-eighth grain, and Lugol's iodine, one minim, daily for the first ten weeks of life, the goitre had not decreased in size. Surgical removal for cosmetic reasons would be considered at a later age. The second patient was a boy of five years, who, for the preceding year, had had a progressive enlargement of the thyroid gland until when he was first examined five weeks previously, his shirt collar could not be buttoned up, and the neck circumference was twelve and a half inches. After treatment with *Thyroideum Siccum*, one-half grain, and Lugol's iodine, one minim, daily the neck circumference had decreased to eleven and five-eighths inches, and the improvement in appearance was most marked. The third patient was a girl, aged nine years, with a soft thyroid enlargement and a neck circumference of eleven and three-quarter inches, who had been brought to hospital two weeks previously, because several relatives on the father's side had had goitres. Her treatment had been started with the same dosage of thyroid and iodine. Dr. Blanch said that in none of the cases had the child lived in a goitrous area at any time.

### Investigation of Hæmorrhagic States.

DR. JOHN COLEBATCH and MISS BETTY WILSON gave a demonstration of the measures that might be necessary in the investigation of a case of hæmorrhagic disease. This was illustrated with a description of the procedures on a demonstration board, and with relevant case histories, microscope slides *et cetera*.

It was stressed that a complete diagnosis required first an adequate history and examination, as was illustrated by a case of almost symptomless recurrent epistaxis which proved to be due to unsuspected chronic nephritis. The clinical report of a case of allergic purpura was presented to illustrate the use of the capillary fragility or tourniquet test.

Descriptions of various laboratory tests were given, with in each case the principles underlying the test, the range of normal results and a list of the conditions in which abnormal results might be expected. These laboratory procedures included full blood and bone marrow examinations, platelet count, bleeding time, clotting time, tests of thromboplastin activity, prothrombin tests and tests for anticoagulants. The use of recently introduced procedures for the differentiation of the hæmophilioid states was illustrated with the clinical and laboratory findings of typical cases of true hæmophilia and of Christmas disease.

### Congenital Heart Lesions.

DR. M. L. POWELL, DR. H. HILLER and DR. WYNN demonstrated patients with congenital heart disease. Dr. Powell showed three infants with patent *ductus arteriosus*. None of these infants had a continuous murmur, but they all had a high pulse pressure. On X-ray examination the hearts appeared enlarged with congestion present in the lung fields. The electrocardiograms were normal. Two of the patients had been operated on, and in one the ductus was 14 millimetres across. The details of this "infant ductus" lesion are being published in a separate article.

Dr. Hiller showed two cyanotic patients. The first was a baby with Fallot's tetralogy, which had been operated on and had undergone a Willis Potts anastomosis one month before. Since the operation the baby had been very well, and no attacks of unconsciousness, which had been frequent before operation, had been noted. The second patient had a pseudotruncus lesion; because of the degree of cyanosis the child was to be submitted to operation in the hope that a pulmonary artery of sufficient size for anastomosis would be found. Dr. Hiller also demonstrated a series of reduction X-ray films of angiocardiograms showing the various patterns seen in many of the congenital heart lesions encountered.

Dr. Wynn showed three patients with coarctation of the aorta. He demonstrated the clinical picture, showed the angiocardiographic picture and discussed the operation. He pointed out that at the Royal Children's Hospital angiocardiography was a routine procedure before operation. The venous route for the injection of the contrast medium had proved entirely satisfactory, and excellent visualization of the coarctation site could always be obtained. In no instance had the technique of retrograde aortography through a brachial artery been required. The operation was not usually undertaken before the sixth or seventh year. Dr. Wynn stressed the importance of clinical examination in cases of this type, with particular emphasis on the absence of or delay in the femoral pulses. Dr. Wynn said that in a number of instances in the Royal Children's Hospital series the brachial artery pressure was not conspicuously raised, and in some the femoral pulses were palpable. In those, however, a marked delay in comparison with the radial pulse was always present. In every case the systolic murmur was louder posteriorly in the interscapular region than over the base of the heart anteriorly. It was believed that that was a most valuable sign in coarctation of the aorta, as it was not found in other forms of heart disease. In 30% of cases of coarctation of the aorta met with at the Royal Children's Hospital there were additional cardiac anomalies, usually aortic stenosis or ventricular septal defect.

### Rhachitic Manifestations.

DR. DOUGLAS GALBRAITH showed three patients to illustrate examples of the varying ætiology of rhachitic manifestations. The orthopædic procedures on two of the children had been carried out by Dr. J. B. Colquhoun.

The first child was a girl, aged three years, who had been born in Melbourne of Lebanese parents. Her condition exemplified rickets due to nutritional defects and inadequate sunlight. Her early diet had been completely inadequate, and she had lived in a dark house, being rarely taken out into the sunlight. When she was first examined, there was definite clinical and radiological evidence of rickets. The results of renal function tests were normal, as were amino-acid excretion and fat excretion. With therapy the rickets rapidly healed, and osteoclasis of both tibiæ on August 11, 1954, gave a successful result.

The second child was a boy, aged two years ,with the typical manifestations of cystine storage disease (Lignac-Fanconi syndrome). His diet had been adequate and had been supervised at an infants' welfare centre. He made normal progress until fourteen months of age, when he went

off food and became cross and restless. Vomiting occurred, and the mother stated that thirst was pronounced. Dr. Galbraith said that the family history was interesting, in that a sister, aged nine years, who was previously regarded as having vitamin *D*-resistant rickets, had recently been shown to have cystine storage disease. There was a history of nephrectomy in a maternal aunt, the maternal grandmother and the maternal great-grandmother. When the patient was first examined the diagnosis of cystine storage disease seemed likely, and the pathology findings, as carried out in the Clinical Research Unit under the direction of Dr. Howard Williams, confirmed that diagnosis. The child had a normal or slightly lowered serum calcium content, a slightly raised serum phosphate content, a raised serum alkaline phosphate content, a lowered serum potassium content, a slightly raised serum chloride and serum sodium content, a lowered alkali reserve, a diminished renal function, a slightly raised blood urea content, a slightly raised serum protein content, increased urinary excretion of amino-acids and cystine crystals in the bone marrow. Dr. Galbraith said that it was interesting that the child developed tetany before treatment was commenced. It was also of interest that although prognosis was said to be poor in such children, there had been great improvement in the condition after treatment with vitamin *D*, 100,000 units daily, calcium gluconate, 60 grains daily, and a mixture of sodium and potassium citrate in a dosage of three grammes of each daily.

The third child was a girl, aged eleven and a half years, with vitamin *D*-resistant rickets of unusual type, possibly due to undetermined renal disease. There had also been an unusually widespread general osteoporosis. She had been well until three and a half years of age, when she was taken to the family doctor because of a limp. A diagnosis of rickets was made, and adequate treatment was commenced. However, there was no improvement in the condition for the next three years, when she was admitted to the orthopædic section of the Royal Children's Hospital. Very slow improvement continued during the next year, but on the correction of the degree of chronic acidosis, great improvement began to occur, and healing was completed within approximately four months. The findings of all renal tests both for glomerular and for tubular function were within normal limits, although the blood urea level was consistently slightly raised. The fat absorption was normal, and the serum electrolyte figures were normal except for a raised phosphate content, a raised alkaline phosphatase content and a slightly lowered alkaline reserve. The condition appeared not to be simple rickets, renal rickets with diffuse chronic renal disease and insufficiency, cœliac rickets or cystine storage disease. It seemed likely to be due to an unusual type of renal disorder with an abnormality in both glomeruli and tubules, although all further tests for cystine storage disease were being made. Medical treatment had consisted of administration of calciferol, 50,000 units daily, calcium gluconate and a sodium citrate and citric acid mixture. Orthopædic procedures for straightening arms and legs had been carried out by Dr. Colquhoun on eight separate occasions with good results. The girl was still dwarfed, and with the apparent fusing of the epiphyses at the lower ends of both femora it seemed unlikely that anything approaching normal growth would occur.

### Morquio's Disease: Osteochondrodystrophy.

DR. L. P. WAIT showed a girl, aged thirteen years, who had been first brought to him seven years previously on account of dwarfism. She was a replica of her mother, who was also a dwarf, her height being three feet four and a half inches. There was no other family history of dwarfism. (The father was five feet three inches in height.) The child had been delivered by Cæsarean section at the Women's Hospital; she appeared to be normal at birth and grew normally till the age of one year, when her mother recognized the similarity to her own condition. The facies and skull of mother and child were perfectly normal in appearance. The findings of a sekeltal X-ray survey of mother and child at the time when Dr. Wait was first consulted had been regarded as characteristic of the osteochondral dystrophy of Morquio. The child's skull was normal. She had flattening of all the vertebral bodies (platyspondilia) and shortening of all long bones, the cortex of which was relatively thick, with "mushrooming" of the lower ends of the femora and both ends of the tibiæ. The proximal ends of the femora were deformed, and the acetabula were abnormal. The proximal ends of the humeri were under-developed. The glenoid cavities were small. The articular ends of the metacarpals and metatarsals were rarefied in contrast with the thick cortex of the shafts of the bones. The carpal and tarsal bones showed uniform rarefaction. The X-ray films of the mother showed similar changes, except in the spine; the vertebral bodies were normal in appearance, but there was very gross scoliosis. Both hip joints showed gross osteoarthritis.

The girl was now aged thirteen years, and a recent skeletal X-ray survey had revealed much the same appearance as already described. The bodies of the vertebræ were now showing a normal appearance. She had developed a pronounced limp in the right limb, for which she was receiving treatment.

Dr. Wait said that on critical analysis, neither the mother nor the child conformed to the clinical appearance described by Morquio. In his description of the entity the neck was short, the spine and chest were severely deformed, the long upper extremities extended to the knees, and there were pronounced *genu valgum* and flat feet, but the face and skull were normal. Neither the mother nor the child exhibited those deformities, but the skeletal abnormalities as shown by X-ray examination were similar to those described by Morquio. Dr. Wait therefore suggested that the patients were suffering from chondrodystrophy with skeletal changes that resembled those described by Morquio but different from those seen in achondroplasia.

### Congenital Vascular Ring.

Dr. Wait's second patient, a boy, aged two years and eight months, had been admitted to hospital in Dr. Wait's care on September 20, 1954, with a diagnosis of "stridor for investigation". It was stated that there had been no symptoms till the child reached the age of five months, when one day, while eating a rusk, he seemed to choke. He was quickly "up-ended" and several pieces of rusk were dislodged from his throat. Since that episode his mother had noticed his breathing to be noisy and "rattly". This varied from hour to hour. A cough developed, but was not troublesome till eleven months before his admission to hospital. Since then he had had severe bouts of coughing, sometimes accompanied by sputum. The cough was sometimes paroxysmal and was usually worse at night. Often his breathing was laboured, and on those occasions the rattle in his chest was more obvious. All the symptoms were aggravated by exercise, by emotional upset and sometimes by eating solid lumpy foods, such as an apple. He had had two attacks of so-called bronchitis, the last attack occurring six weeks prior to admission to hospital. On examination he was found to be a healthy boy with obvious stridor. His cough was "brassy". There was moderate chest retraction. No heart abnormality was detected. His blood pressure was 110 millimetres of mercury, systolic, and 70 millimetres, diastolic. His femoral pulses were strong. It was considered that he could have a congenital vascular ring as a cause of his symptoms. Radiological investigations with swallowing of a barium bolus showed an indentation and narrowing of the œsophagus on both right and left sides in the upper third. A tracheogram showed a narrowing of the trachea on the right side and posteriorly, similar in position to the narrowing found in the œsophagus. An aortogram showed a right-sided aorta with the vessels of the left arm and left side of the head passing posteriorly to the œsophagus and trachea. There was an apparent diverticulum of the aorta just distal to the origin of the vessels to the right side of the head and right arm. The *ligamentum arteriosum* probably arose from that structure. The last-mentioned structure completed the ring which enclosed the trachea and œsophagus.

### Dermoid Cyst of Posterior Cranial Fossa.

Dr. Wait's last patient, a girl, aged nine months, had been admitted to hospital on September 28, 1954. She had been regarded as a "feeding problem" since birth. There were frequent bowel actions every day. In the past four weeks she had had bouts of vomiting, for which she was given "Eumydrin", but that was discontinued after three days as it appeared to give her the "shakes". She was tried on various milk feedings without apparent improvement in her condition. When aged four and a half months she had a febrile illness for which she was given "sulpha" tablets. Fever recurred two weeks and four weeks later. In the last attack she was given "sulpha" drugs and "Chloromycetin". For the past four months she had screamed practically every night and sometimes during the day. The parents were unable to comfort her. She had never made any attempt to sit up.

She was finally referred to the out-patient department on account of tremor and her inability to move her limbs. A lumbar puncture was performed, as she was thought to have polyneuritis. The cerebro-spinal fluid contained four polymorphonuclear cells and 11 lymphocytes per cubic milli-

metre, a protein content of 80 milligrammes *per centum* and a sugar content of 40 to 50 milligrammes *per centum*.

On admission to hospital she appeared to be a miserable apprehensive baby with generalized muscular atonia but no paralysis. Over the occipital protuberance a small red raised lesion was noted, and on closer inspection a fine tuft of hair was seen to be protruding through a small sinus. Examination of the fundi showed gross bilateral papilloedema. The head circumference was 18 inches. The fontanelle was not tense. She was regarded as having an intracranial dermoid tumour.

An X-ray examination of the skull showed a deficiency in the occipital bone which confirmed the clinical diagnosis. At operation a small intracranial dermoid tumour was discovered, but it was difficult to identify the cyst wall, as it was imbedded in a multilocular abscess mass situated mostly in the left cerebellar hemisphere. However, Dr. R. S. Hooper was able to excise the tumour and the abscess. Her post-operative course was uneventful. She was now a contented, happy baby. She played with toys and appeared, at the present, to have made an excellent recovery. Culture of material from the abscesses failed to produce any growth of organisms. The protein of the cerebro-spinal fluid rose to a level of 330 milligrammes *per centum*. On the patient's discharge from hospital it had fallen to 130 milligrammes *per centum*.

## Out of the Past.

*In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.*

### DISEASES OF THE NATIVES.

[From "The History of New South Wales, including Botany Bay, Port Jackson, Parramatta, Sydney and All its Dependencies from the Original Discovery of the Island with the Customs and Manners of the Natives and an Account of the English Colony from its Foundation to the Present Time: by George Barrington,[1] Superintendent of the Convicts", London, 1802,][2]

In all ages diseases have been the companions of mortals and the natives of New South Wales have their share: most nations have their cures and all attempt to cure the maladies by which they suffer. A pain in the belly they cured formerly by breathing on the hand to warm it and applying it to the part affected, singing a suitable song to the occasion and keeping the mouth near the affected part, frequently stopping to blow on it and make a noise after blowing like the barking of a dog: but our settling there rendered this useless as tincture of rhubarb saves this trouble.

In 1789 a disorder in appearance like the smallpox raged with incredible violence. Some Gentlemen of the Colony took a native to the beach to find his former companions and so much did his agony and expression affect them that it will never be effaced from their minds: with an anxious eye he searched round every cove: no print of human foot was to be seen on the sand; the caves in the rocks were now the tombs of the dead, and not one living soul was to be seen—all had flown—in the silent agony of grief he lifted up his hands and then exclaimed "all dead! all dead!" and in mournful sorrow again hung down his head, nor did he lift it up again during the excursion. This poor fellow at length suffered the fate of his companions, for on some of the natives being brought to the Colony he caught the disease and died. With such violence did this disorder rage that the country seemed desolate, and one whole tribe of natives was swept off except three persons.

It is remarkable that though Sydney was full of children at that time and they visited those natives that were ill, not one caught it.

Those on the coast have a disorder very much like the itch, sometimes it is very general: in 1791 it raged so much that many came to the Colony in a shocking state and every native seen had it with more or less violence. The venereal disease, there is every reason to imagine they were not ignorant of before they knew us, but if they were, our arrival will account for its appearance shortly after, though every care on the part of the Governor was taken to prevent it. However, an intercourse between the people soon took place, a native woman had a child by a white man: but seeing the child incline more to white than the colour of her other children, she held the little unfortunate over the fire in the smoke rubbing dirt and oil over its body to give it the wished-for hue. When they have a pain in any part, they tie a ligature very tight round the part and thus stopping the circulation of the blood give ease to the part affected.

[1] George Barrington was a famous pickpocket who was sentenced in 1790 to seven years' transportation. His behaviour in the colony was exemplary: he was given many positions of trust and was appointed Superintendent of Convicts and later Chief Constable. He retired on a pension in 1800 and died insane in 1804. The above and several other similar histories were published under his name, but in every case he repudiated the authorship.

[2] From the original in the Mitchell Library, Sydney.

## Clinico-Pathological Conferences.

### A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on November 16, 1954, the medical superintendent, DR. NORMAN ROSE, in the chair. The principal speaker was DR. W. L. CALOV, Senior Honorary Physician at Sydney Hospital.

#### Clinical History.

The following clinical history was presented.

The patient, a retired seaman, aged sixty-one years, came to hospital complaining of breathlessness and pain in his chest for a month and died a fortnight after admission. Even in retrospect it is difficult to find any clue to the diagnosis in the clinical notes. The only reason for presenting such a puzzling case is that the disease which killed the patient is by no means a rarity, and it is a curable one.

Six months before admission to hospital the patient was referred to the pulmonary clinic for investigation of an abnormality detected by mass radiography survey. Atelectasis of the lower lobe of the right lung of undetermined cause was found. For the last month he had had severe pain in the right side of his chest, worse with coughing or breathing deeply, a dry cough, dyspnœa with minimal exertion, orthopnœa and some swelling of his ankles. There had been no hæmoptyses. There was anorexia. His bowels acted regularly once a day, and his motions were normal in colour and consistency. Urine was passed thrice at night and twice during the day, without dysuria. Apart from some deafness, a right facial palsy and poor vision caused by a fractured skull thirteen years before, he had no other symptoms.

Previous illnesses included "dropsy" eleven years earlier, an injury to the right side of his chest six years earlier, and "waterlogged lungs" four years earlier. There was no relevant family history. He was a single man living in poor circumstances on a poor diet.

On examination he was flushed, apyrexial and dyspnœic, with a pulse rate of 102 per minute and a respiratory rate of 34 per minute. He had œdema of the legs, ascites, a raised venous pressure and an enlarged liver. The heart was enlarged and fibrillating. The blood pressure was 95 millimetres of mercury, systolic, and 55 millimetres, diastolic. On the right side of the chest there were signs of an effusion. The left side was clear. No abnormalities of the central nervous system were detected apart from the results of the injury mentioned above. The ankle jerks were not elicited, the knee jerks and plantar responses were normal. The urine had a specific gravity of 1024 and no abnormal constituents.

X-ray examination of the chest showed a huge pleural effusion on the right side and mediastinal shift to the left.

Electrocardiographic examination showed a low voltage tracing, left axis deviation, auricular fibrillation and premature ventricular contractions.

No organisms were seen in or grown from the pleural fluid.

The blood contained 8·7 grammes of hæmoglobin per 100 millilitres, and the white cell count was 14,300 per cubic millimetre (80% were neutrophile cells, 16% lymphocytes, 2% eosinophile cells and 2% monocytes).

The provisional diagnosis was congestive cardiac failure, and he was treated with digitalis, "Mersalyl", vitamin *B*, ammonium chloride and a salt-free diet. There seemed to be some improvement for a few days, but then the œdema increased, becoming particularly prominent on the right side of the abdomen wall and chest. There was no fever. He died suddenly.

### Clinical Discussion.

DR. N. H. ROSE: As the composer of this clinical history states, this is the record of a patient who suffered from a disease which is not uncommon. By using the seventh sense it might be picked. I will now ask Dr. Calov to discuss this difficult and interesting case.

DR. W. L. CALOV: The story starts with the unpromising statement that even in retrospect it is difficult to find any clue to the diagnosis. Not having the chance of retrospection and only having a mere crystal ball into which I have gazed for several hours, I have not yet obtained a clue.

We start with this patient's age. At sixty-one he was at an age when he might have almost anything, and some of the findings we read of later are not altogether surprising. Another point about it is that he was a retired seaman. I always believe it is an important thing to bear in mind the patient's occupation, and in the case of sailors there is that old saying that "sailors don't care", and sometimes we find they do not care to the extent that they become infected with syphilis. So syphilis is a thing that I would always be inclined to bear in mind when I read about a retired seaman. By retired seaman I think of a merchant sailor rather than a sailor of the Navy. An ex-naval man is much more likely to be in good health than a merchant sailor because of the discipline and the way in which naval men are looked after.

Further on we find that the disease which killed this fellow is by no means rare and is curable. This stumps me immediately, because after reading through this the obvious diagnosis is bronchogenic carcinoma; but as we have learned before, bronchogenic carcinoma is by no means curable. That makes it very difficult. In fact it makes it impossible. Lower down we find this man had an atelectasis of the lower lobe of the right lung of undetermined cause. Well, it is a wonder that they did not ascribe some cause to it. The commonest cause of atelectasis of the lower lobe of the right lung—collapse would be a better word—is obstruction to the bronchus by a carcinoma. It could be obstructed, of course, by other tumours such as a simple adenoma; it could have been caused by the injury which we find he had several years ago, an injury to the chest; it could have been caused by infection, and the most likely infective causes of collapse of a lower lobe would be tuberculosis and syphilis. It could also have been caused by an old pneumonia in which the lung failed to reexpand. It could have been caused by a foreign body, or it could have been caused by intraabdominal disease. The intraabdominal disease might have been a huge tumour, ascites or, what is more likely, intraabdominal infection, suppuration such as might result from ruptured ulcer, or perinephric abscess. These things are possibilities, but they do not fit in very well with the later findings.

This man had severe pain in the right side of his chest, worse with coughing or breathing deeply—characteristic of almost any pain in the chest except cardiac pain. This implies that the pain resulted from some involvement of the pleura. That is the first proposition. It could be simple pleurisy or hæmorrhage into the pleural space—I remember a case presented here once in which there was recurrent hæmorrhage into the pleural space from an aneurysm which used to leak, and the man used to get dreadful pain from this—but I do not think this is the kind of pain produced by intermittent hæmorrhage, because apparently the pain was persistent. It could be due to involvement of the pleura in a neoplastic process. It could be due to some involvement of the mediastinum either by glands or by suppuration such as may result for instance from rupture of the œsophagus. Mind you, I am saying quite a few fantastic things which do not really fit in, so far as I can see, with the later story. It could result also from a massive collapse of the lung with indrawing of the parietes or with mediastinal shift, both of which will cause considerable pain. Or it could have been due to some involvement of the parietes, say the ribs or the muscles of the chest wall, and they could have been affected by newgrowth or by distortion as I mentioned earlier.

He had a dry cough. Well, he had some kind of a disease inside his chest, and it is not altogether surprising that he had a cough. Years ago we were taught about all sorts of different kinds of cough, but now you find that many of these symptoms to which we used to attach a great deal of importance do not have the same significance to the modern generation. They merely say that this patient had a dry cough. One of us older people would probably have described that cough more minutely if we had thought it to be of any great significance. All that we know is that this fellow had a cough, and he did not spit with it. I think we can say with regard to that cough that it probably was not due to infection, for if it had been due to infection, the probability is that there would have been some spit. He had dyspnœa with minimal exertion. We imagine he should have dyspnœa—he had a chest full of fluid, and his heart was not functioning very well either.

He had no hæmoptyses. That is important in view of the collapsing of his lower lobe, because if he had a carcinoma you would think, or hope, that he might spit up a little blood and give you some guide to the diagnosis. But he did not, and that, combined with the statement in the opening paragraph, rules out carcinoma. He had anorexia merely because he was a sick man, and I do not think that symptom is significant. He had frequency of micturition at night, passed urine three times; but then I suppose it is not unusual for a man of sixty-one to get up once or twice at night, and this fellow was a sick man with a cough and a pain in the chest. I suppose he used to wake up pretty frequently; and when a man wakes up at night, round sixty-one at any rate, he very often feels inclined to pass urine. That is probably what happened to this fellow, and I think we can rule out his nocturnal frequency. He passed his urine only twice during the day, you notice—that is because he was retaining a lot of fluid, and it was not going out through his kidneys as it should have been. So I think he did not pass a great deal of water at all.

With regard to the fractured skull and the defects caused by that, I do not think we need say much about it. If you had a full discussion of everything this man had, we should be here till midnight. It is reasonable to believe that these signs resulted from the fractured skull. He had dropsy eleven years ago and "waterlogged" lungs four years ago. I suppose that this fellow had a bad heart for a long time, and off and on he had gone into failure. I do not know what those "waterlogged" lungs were, whether he had bilateral pleural effusion or whether he had pulmonary œdema. It might make it easier if we regard its as pulmonary œdema, which would be a more likely thing.

The injury to the right side of the chest six years ago could have been the cause of the collapse of his lower lobe. I do not know whether it was or not. Remember that he found out about his collapsed lobe almost by accident at a routine X-ray examination, and it may be that he had been getting around with collapse of his lower lobe for many years.

He was a man living in poor circumstances on a poor diet. I do not know quite how much importance is attachable to that. I do not think it was directly concerned in his death, but it could have been responsible at any rate for the absence of his ankle jerks. I do not suppose he was ingesting a large quantity of vitamins or of good food, good protein and so forth, and it may well be that this fact contributed to some extent to his illness.

He was dyspnœic and he had a respiratory rate of 34 per minute, which is out of proportion to his pulse rate of 102 per minute, and this indicates respiratory distress and suggests to me that there was some respiratory disease or respiratory involvement.

Œdema of the legs, ascites, raised venous pressure, enlarged liver, enlarged and fibrillating heart, all indicate that he had congestive cardiac failure, and allow no doubt that whatever he died from he did have congestive cardiac failure. I am inclined to doubt that the heart was enlarged—it was apparently enlarged because his right thorax was full of fluid and the negative pressure in his left hemithorax pulled the heart and mediastinum over; the apex beat was no doubt felt wide out, and the conclusion reached that he had an enlarged heart. It may well be that he did not have an enlarged heart at all, and I think from the appearance of the X-ray films that the heart was not grossly enlarged. His blood pressure was 95 millimetres of mercury, systolic, and 55 millimetres, diastolic, which fits in with heart failure. On the right side of the chest there were the signs of an effusion, and the left side was clear. This is interesting, because congestive cardiac failure can cause an effusion on one side only, usually the right side for some reason which I will not attempt to discuss; but when one

hemithorax fills right up and that filling is due to congestive cardiac failure, you usually expect to find at least some fluid on the other side. So, I believe that the effusion was not entirely due to congestive cardiac failure. There was some other cause for it.

There were no abnormalities in the central nervous system except that the ankle jerks were not elicited. Well, ankle jerks are sometimes difficult to elicit; and we must remember that this was an old fellow on a poor diet, and he may have had arterial degeneration as well, which would be responsible for some degree of ischæmic neuritis, and so it is not altogether surprising that he had no ankle jerks. His pupils were equal and active, and so I do not think tabes comes into it.

Now, X-ray films of the chest showed huge pleural effusion on the right side and mediastinal shift to the left. Could we have the films now, Mr. Chairman? The early films showed a shift of the heart and mediastinum to the right, which resulted presumably from collapse of the lower lobe.

DR. R. JENKIN: This is the first film taken during an anti-tuberculosis survey. It shows a left lung which is apparently clear and an opacity at the middle and lower zone of the right lung. The trachea is moved across to the right, there is a slight shift of the mediastinum to the right, an old fractured rib can be seen, and apparently a metallic foreign body is present. That film was taken about six months before he died. A week after the first film he came here for investigations. Antero-posterior and lateral views of the chest showed the same thing—fractured rib, clear left lung, opacity in the right lower lobe, and trachea shifted to the right. In the lateral view can be seen a calcified node and pleural thickening or fluid.

The next step in investigation was a "barium swallow". It showed no great cardiac enlargement, it showed the trachea deviated to the right side, an opacity in the right lower zone with a concave border towards the mid-line and some increased opacity apparently of the lung substance itself. The next step was a right lower lobe bronchogram, which confirmed that there was some collapse of the right lower lobe, the opacity below the collapsed lung being due to fluid or pleural thickening. At that stage our investigations stopped. He next came in about four months later showing this picture. The trachea is in the mid-line, with slight shift to the left of the mediastinum and almost complete opacity over the right lung. Another film taken seven days later shows further opacity on the right side and further shift of the heart and mediastinum to the left. The last film shows even further displacement, and now there are slight congestive changes in the left lung.

All the honorary radiologists have seen these films, and they are all puzzled. Certain suggestions have been made, they have seen the history and know that it is a curable condition, and they know that the patient was apyrexial. The general opinion is that it could be either a hæmothorax—an old hæmothorax with compression collapse of the right lower lobe—or chronic empyema. They base that on the original films, which show what appears to be an effusion at the base of the right lung *plus* a shift of the mediastinum over to the same side, which occurs most commonly with carcinoma or quite often with chronic empyema. Whether it is primarily a chest lesion or a subphrenic one we are unable to say.

DR. CALOV: My reading of those films is that the man had a collapsed lower lobe to start with and later on developed an effusion. That is all I can say about it. An electrocardiogram showed a low voltage tracing with left axis deviation. I should imagine that if the electrocardiogram had been taken earlier it might have shown something a little different from that so far as the axis deviation is concerned, because the heart has only recently been pushed, or rather pulled, over to the left. He had auricular fibrillation and premature ventricular contractions.

Now no organisms were seen or grown from the pleural fluid, so I think that rules out the suggestion that it was an empyema. But the pleural fluid was bloody. When I see blood-stained pleural fluid and I think it is genuine—that is, blood-stained before the needle was stuck in—I think of carcinoma, and my mind just will not go beyond that and I cannot make it. If it is not due to carcinoma it could be due to leaking from an aneurysm, but I do not think that occurred in this case; it could be due to inflammation, but you would expect organisms or polymorphonuclear cells in the pleural fluid. It could be caused by neoplasm of the pleura. Neoplasm of the pleura is very rare and would not cause collapse of the lower lobe; so our difficulties increase as we go along. His blood count fitted in with either an inflammatory lesion or neoplasm. I would say that the low hæmoglobin value, not terribly low, and a polymorphonuclear leucocytosis fit in with carcinoma or with an inflammatory condition. The results of Wassermann and Kahn tests are negative. I do not suppose that rules out syphilis altogether, and I still have an uneasy feeling that he might have had syphilis and that the collapse of a lobe might have been due to a gumma, but you would expect him to have spat something at some time or another. Syphilis can do all sorts of queer things, even in the lungs, and, of course, it is not common to diagnose it clinically in the lungs, and I think it is very rare to see syphilitic lesions in the lungs after death.

Provisional diagnosis was congestive cardiac failure, and I do not doubt that that was quite a correct diagnosis. But I do not think it explains the extraordinary X-ray appearances and the extraordinary history. Now we find the œdema increased, becoming particularly prominent on the right side. I suppose that is because the fellow lay on that side. I do not suppose it was due to any other cause. It is the result of gravity. He lay on that side because he had a better chance of breathing reasonably well if he was turned towards his right side, and that is the way the fluid would run.

He died suddenly. Well, there are several ways in which he could have died suddenly. He could have had a coronary occlusion, an infact in the other lung or an embolus in the brain from his fibrillating auricle, and there are other things you can think of. He was a very sick man, and it does not make any difference to the diagnosis whether he died suddenly or not. He had enough disorders there to make him die suddenly.

As far as the diagnosis is concerned, as I mentioned earlier, the wind was taken out of my sails by the first paragraph of the story. So I am afraid that the diagnosis is a bit nebulous. In fact there is no diagnosis at all so far as I can see. I have written down here several things I do not think it could possibly be. The first of those is carcinoma because it is eminently incurable. Syphilis—I still feel it might be. I am not going to discard that entirely. So if he does turn out to have syphilis, you can say I said he might have had it. He did not have tuberculosis. I think that is pretty sure; it was not tuberculosis that caused all this. You would have thought he would have brought up some spit, had some night sweats, some fever. I do not think it could have been an endothelioma of the pleura. It is rare, but I suppose it is possible, but, if so, it did not cause all the other things. He did not have an abscess of the lung. He would have been pyrexial, and after those months surely he would have coughed something, the abscess would have burst into a bronchus somewhere, and surely, also, he would have had some organisms in his pleural fluid as well as pus cells. He had none of the other signs of abscess of the lung—there is no mention of a foul breath or of clubbing of the fingers. I do not think that it could have been a hydatid. With a hydatid one would have expected to see a rounded shadow in one of the earlier films. Anyway, it would be extremely rare for hydatid to cause collapse of a lobe of a lung. Hydatid does not usually press on a bronchus and cause lobar collapse.

Foreign body: there appears to be a foreign body there, but whether it is right in the lung substance or not I do not know; whether it is in the lower lobe bronchus I do not know. If it was in the lower lobe bronchus, it could cause collapse. It may well have been there causing collapse, but it could not be responsible for the rest of the trouble. He could have had an adenoma of the bronchus, but it could not have caused all that pleural effusion. It could have caused collapse of the lobe, and the collapse, with its pull on the mediastinum, could have caused pain; but I do not think that adenoma can be regarded seriously. It could be a lipoid pneumonia. Lipoid pneumonia occurs more frequently than we realize. There have been several cases recently, two from the pulmonary clinic, which we regarded as tuberculous, and when we sent the patient for a thoracotomy and removal of the affected segment of the lung it turned out to be lipoid pneumonia. There is no history of the use of paraffin or inhalation of oily liquids, but I suppose nobody thought of lipoid pneumonia and therefore they would not have thought to ask him about it. You do not usually think to ask the patient about these things till afterwards. So I just put that in for good measure, just to show you I remembered there is such a thing as lipoid pneumonia. Constrictive pericarditis has a number of things against it, but not the radiographic appearances. But there is no mention of *pulsus paradoxus*, and there is no evidence of pulsating liver. All the signs could result from constrictive pericarditis. You would like

to see calcification, but this is not always found, at least none that is apparent radiographically.

The final thing that entered my mind, a mind beset with all sorts of horrible and uncomfortable thoughts, was rupture of the œsophagus. But it cannot be that. There is no evidence of surgical emphysema, which there should be if he had a ruptured œsophagus. I merely mention it, without ruling it out entirely, but I do not think it is very likely. There they are. No diagnosis made, but a few suggestions, and I have not the slightest hope that any of them will be the correct one.

DR. ROSE: Dr. Calov, I am sure, has mentioned every possible medical condition which can cause this picture. There are also surgical conditions, of course, which could be mentioned as causing a condition like this. We would like someone to stand up and emphasize what he thinks would be a more likely diagnosis. I judge from what Dr. Calov has said that without the first paragraph he would put all his money on carcinoma of the lung as a primary diagnosis, but that has been excluded. Would somebody like to discuss this diagnosis?

DR. C. B. HUDSON: I think there are two most striking features about this case. One was that the patient obviously had some pulmonary disease, and the other was that he had cardiac disease. Now, it seems that he may have had cardiac disease for some years, but the examination does not indicate what type of cardiac disease he had. I think that anybody that was a sick as he was might easily develop auricular fibrillation and congestive cardiac failure just before he died, but there is some indication that he may have had cardiac disease for a long time. There is not any evidence that he had ever been hypertensive. There is no evidence of valvular disease. I think that Dr. Calov's suggestion of a vitamin deficiency must be considered. He might have been alcoholic and been on a poor diet and have had beriberi. I noticed he was given vitamin *B*. If that was given in adequate amounts parenterally, that should exclude beriberi heart, but it may have been too late. The cardiac picture is, I think, secondary and not the actual cause of his death.

From the pulmonary point of view it appears that he had some lesion that caused the lower lobe of his right lung to collapse and caused a cough, and it was apparently a benign lesion, because we are told it was curable. Foreign body, as Dr. Calov mentioned, is a possibility, but I think that a bronchial adenoma could explain this story. He developed pulmonary collapse, he developed, perhaps, some low-grade infection in the collapsed lung, and his heart was in poor state. The pleural effusion could be due to the combination of a low-grade infection and congestive cardiac failure. So I would put my money on a benign pulmonary neoplasm.

DR. ROSE: Dr. Ferguson, would you care to say something? Dr. Ferguson is from general practice, and as this is a curable disease, I think we are likely to get the diagnosis from a person like Dr. Ferguson.

DR. D. A. FERGUSON: In general practice we do not often meet a thing like this, and if we do we send the patient into hospital and let them make the diagnosis. I cannot help remarking on the marked discrepancy between the amount of fluid in the right side of the chest and its absence in the left side. One wonders whether there was any venous obstruction to account for this. However, there do not seem to have been any of the usual symptoms of pulmonary œdema—cough, blood-stained sputum or anything like that. I feel he must have had some sort of neoplasm in his chest.

DR. ROSE: Dr. Read, would you care to say something?

DR. F. READ: Being late in arriving, I hoped I had come in just in time for the answer. The fact that he had atelectasis of undetermined cause may, of course, be a red herring. We have not actually considered the causes of auricular fibrillation, which, I guess, would be thyrotoxic or arteriosclerotic. I only bring this in to complicate the picture, because we know that the disease which killed the patient is by no means a rarity and is a curable one. Here is a man who has auricular fibrillation and who is in a state of failure. I notice that the notes say the left lung was clear, but it does not look clear in the film, and I feel sure that there must have been some signs of left ventricular failure present. I feel we have here a picture of a red herring in the lung, an undisclosed cause of auricular fibrillation with failure and very likely, I think, an added complication of vitamin *B* deficiency.

DR. ROSE: Is there any further discussion? It does strike me that not enough emphasis has been placed on what I consider to be the most important of the special tests done, the vagueness of the findings in the pleural effusion. I somehow sense that by careful examination, not by sending specimens to the pathologist, but by looking at it with the naked eye and doing a few simple tests, the diagnosis may have become apparent. I am surprised that none of the clinicians has queried the naked eye appearance of the effusion. You see there is a note only of whether it was sterile or otherwise. It will be interesting to see whether the diagnosis may have been made by a more thorough investigation of that fluid. There should be some interesting discussion following the autopsy report which I will now ask Dr. Palmer to give.

## Autopsy Report.

The following report was made available by Dr. A. A. Palmer, and slides were shown.

Autopsy was performed eighteen hours after death. The body was that of a man of about sixty in a good state of nutrition.

*Thyroid.*—The thyroid was a little enlarged, but appeared normal.

*Pleural Cavities.*—The right pleural cavity contained about 100 ounces of thick dark reddish-grey fluid. Dense adhesions were present from the apex and anterior surface of the lung.

*Right Lung.*—The right lung was completely collapsed.

*Left Lung.*—The left lung weighed 28 ounces (normal 15 to 21 ounces) and was congested.

*Heart.*—The heart weighed 18 ounces (normal nine to 13 ounces). The pericardium was adherent over most of the surface and was calcified in one area anteriorly and in another region posteriorly. (The weight includes adherent pericardium). The mitral and tricuspid rings were a little dilated, admitting three and four fingers respectively. The valves and chambers otherwise appeared normal. The coronary arteries were remarkably free from atheroma.

*Liver.*—The liver weighed 65 ounces (normal 50 to 60 ounces). It was enlarged and its capsule thickened and whitish. The superior surface of the right lobe was firmly attached to the diaphragm. When these adhesions were cut, an abscess was seen in the liver measuring four inches by three inches and filled with thick white pus. A zone of chronic venous congestion surrounded the abscess. The remainder of the liver appeared within normal limits and showed no sign of chronic venous congestion.

*Spleen.*—The spleen weighed 18 ounces (normal five to six ounces). It was enlarged. The cut surface was rather firm. The splenic artery was calcified.

*Alimentary Tract.*—The œsophagus, stomach and small intestine appeared normal. In the mucosa of the large intestine there were numerous ulcers with shaggy floors.

*Other Organs.*—The kidneys, bladder, prostate, testes and brain appeared normal. The left adrenal appeared normal. The right adrenal was not found.

The microscopic sections confirmed the diagnosis of amœbic colitis and amœbiasis of the liver. There are very numerous amœbæ in the ulcers in the colon and in the abscess.

## Pathological Discussion.

DR. A. A. PALMER: In summary then the heart was somewhat dilated but not greatly enlarged, the pericardium was adherent and in two places calcified, there were ulcers in the large intestine, an abscess in the liver and very extensive right pleural effusion in which the fluid was thick and dark, greyish-red, and the right lung was completely collapsed.

The diagnosis seemed to be amœbic colitis, with liver abscess and pleural involvement on the right side. There is not, you will notice, a lung abscess, but only an abscess in the pleural space with collapse of the lung. In case you might imagine that this is an extremely rare complication of amœbiasis, here is an X-ray picture from Manson-Bahr's text-book showing a similar state of affairs. Amœbic abscesses occur in about 2% to 5% of male Europeans affected with amœbiasis, and the lung is affected in some 5% to 10% of cases in which there is an amœbic abscess of the liver. The sequence of events, of course, is that the liver abscess approaches the diaphragmatic surface, and the diaphragm then becomes adherent over that region as shown in these pictures. In over 80% of liver abscesses the lesion is in the right lobe. Usually the lung becomes adherent to the diaphragm, and the abscess spreads into the lung. But the formation of a pleural effusion and an amœbic

pleurisy occurs occasionally; in this case perhaps the associated heart failure provided some fluid to keep apart the lower surface of the right lung and the diaphragm, and consequently a lung abscess did not form.

DR. ROSE: Anyone who has attended many of these clinico-pathological conferences will always keep in mind amœbiasis and bacterial endocarditis, which seem to appear on many occasions.

DR. W. K. INGLIS: Mr. Chairman, I should like to draw attention to a comment you made with regard to the naked eye appearance of the material which was at present in the pleural sac. Not infrequently the appearance is quite striking. My attention was first drawn to that some thirty years ago, when Mr. James Young, a technician in the department of pathology, came with a test tube of material which had been coughed up, and he looked at it and said: "That patient has an amœbic abscess of the liver." It is sometimes, not always, quite striking. The description was given of it being quite thick and greyish-red. Had this been recorded in the notes, the possibility of the diagnosis might have been brought to mind.

DR. H. K. WARD: I am interested to know at what stage the author of the case history considered the disease curable.

DR. H. M. WHYTE: Do I have to defend that statement very vigorously, sir? Perhaps the main reason I put that in was that I did not expect anyone to diagnose it, and I think it is nice to know at the beginning that you are not expected to, rather than to get to the end and find you cannot. The other reason is that I do not believe in putting a too puzzling case up for clinico-pathological discussion unless there is some lesson to be learned from it. This is an unusual presentation of a disease which, even if not strictly curable in its later stages, would have been handled very differently if the diagnosis had been made during life.

DR. WARD: Should the diagnosis have been made during life?

DR. WHYTE: I do not know about "should have been". But I think the answer lay in the proper examination of the fluid. Time and time again this sort of thing is found in the hospital records, whose compilation is mostly left to a junior resident, an inexperienced resident, who is seeing pleural fluid perhaps for the first time and does not recognize that there is anything very unusual in it. The hospital notes give no other description than that shown in the clinical notes. I think a more experienced observer would have investigated it more fully. It happens in other conditions, too, that the diagnosis can be picked up by simple clinical means; and yet, in retrospect, in looking through the records, no clue can be found to the diagnosis.

DR. R. B. WILES: With respect to looking in retrospect for the diagnosis, there is one thing of which I was not quite sure, and that was the question of the bronchial tree as shown in the bronchogram, which I think filled more or less completely and showed only the alveolar tissues to be collapsed. I wondered whether the radiologists could tell us whether that is typical of collapse caused by pressure from outside rather than by blockage in the tree. In the early radiographs there was a wedge-shaped formation in the lower front part of the thorax, and the lung above it did not seem to be greatly disturbed. I wondered whether there could be seen in this a clue to earlier diagnosis and whether further investigation of this lesion might have revealed the cause.

DR. JENKIN: I have seen several instances of compression atelectasis which have given the same appearance in the bronchogram, with the lack of alveolar filling at the periphery. In the cases I have seen this happened with compression and not with absorption atelectasis.

DR. WHYTE: As Dr. Calov has probably had more experience of amœbiasis in his career in New Guinea than most of us here, perhaps he could tell us something of this disease.

DR. CALOV: There are a couple of things I would like to say in self-defence, Mr. Chairman. First of all, this patient had amœbic ulceration. Well, I should be inclined to doubt that he had ordinary brown stools. I should be surprised if he did not pass some blood now and then and if his stools were not occasionally foul-smelling. Furthermore, there is no record of the patient having pyrexia. I have never seen amœbic hepatitis without pyrexia. It might be only very slight; but once amœbiasis is complicated by hepatitis, there is, in my experience, always fever. Of course it is quite right what several people have said about the fluid. Somebody should have seen that there was something wrong with that fluid, that it was not just blood-stained and no organisms were to be found. It is interesting that he did not have an abscess of the lung. When an amœbic lesion ruptures into a bronchus, the pus is coughed up. I do not know anything else which gives pus like that.

It was a most instructive case and illustrates how helpful and important simple clinical observations accurately recorded might be.

DR. ROSE: Dr. Calov has made a very good job of an undiagnosable case, and we congratulate him.

**Diagnosis.**

Amœbic colitis with liver abscess and pleural involvement.

# Correspondence.

## HIATUS HERNIA.

SIR: The clinical flavour of the recent article by Dr. Hall and Dr. Newton will be appreciated by those practitioners who are interested in this subject. In a slightly larger experience I have encountered two problems which do not appear to have been prominent in their cases.

Firstly, four patients in my series had a transthoracic repair of a hiatus hernia to which their symptoms were attributed. Shortly after this operation the features of pyloric obstruction became so marked as to require gastric resection for the obstructing pyloric ulcer. I take it that the increased intragastric pressure made itself evident first at the diaphragmatic hiatus, whereas the basic lesion was pyloric and merited prior consideration. The abdomino-thoracic approach provides a ready answer to these combined lesions.

Secondly, three patients in my series had well-marked cardiospasm in association with the hiatus hernia, so that a cardio-œsophageal myotomy of the Heller type was required. The results of these operations are encouraging.

Dr. Hall and Dr. Newton are to be complimented upon their careful approach to the assessment of these patients, an attitude with which I most cordially agree.

Yours, etc.,

149 Macquarie Street, Sydney, Undated. K. W. STARR.

## A DISCLAIMER.

SIR: I would be obliged if you would publish a disclaimer on my behalf concerning an article published in *The Sun-Herald* on April 3 reporting work on PAS in rheumatoid arthritis. The details were taken from THE MEDICAL JOURNAL OF AUSTRALIA without my authority.

Yours, etc.,

185 Macquarie Street, Sydney, April 5, 1955. BRIAN HAYNES.

## LUNG CANCER AND SMOKING.

SIR: I have read with interest the letter from Dr. B. A. Stoll. Similar letters from medical practitioners have appeared in various places, some containing even more specious arguments in mitigation of what is now known to be a dangerous habit.

That smoking is a most important factor in the promotion of squamous cell carcinoma of the lung is established beyond any doubt, and that the serious mortality from this has not yet reached its peak is equally certain. It will be most unfortunate if our profession, many of them victims of the habit, do not take all reasonable steps in their power to put an end to it. Even if, for those of us who have smoked since we left school, it is too late to take any effective action, abolition of smoking for those who have not yet acquired the habit will surely save many from a relatively unpleasant and commonly premature form of death.

Though it is in the case of lungs that the relationship of smoking to carcinoma is established beyond doubt, I am certain, after twenty-five years of practical interest in the

subject, that the risk of cancer of the mouth, nasal sinuses, pharynx, larynx, œsophagus and stomach are all enhanced, and that their incidence would be diminished by abolition of the tobacco habit.

It is clear from the statistical evidence that smoking is not the only factor involved. Time relationships appear to absolve smoke from coal combustion, but most serious suspicion falls on the exhaust products of internal combustion engines, especially those consuming diesel fuel. Traffic congestion in cities and the substitution of diesel buses for electric trams may well prove to be important factors, and it is a matter of urgency to determine whether this is the case or not; for if the facts are established, it may be necessary to ban all petrol and oil vehicles from congested areas and replace all diesel-powered public road transport with electric traction.

It is the duty of the medical profession to promote and preserve health regardless of any other considerations or interests that may be involved. As soon as it is reasonably well established that any substance is a carcinogen, we must spare no effort to ensure that its presence in any circumstances which may endanger the public is eliminated, whatever may be the cost.

Yours, etc.,

J. ORDE POYNTON.

The Institute of Medical and Veterinary Science,
Adelaide,
April 6, 1955.

# Congress Notes.

## AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE following notes relate to the Australasian Medical Congress (British Medical Association), Ninth Session, which is to be held at the University of Sydney from August 20 to 27, 1955.

### Patron.

His Excellency the Governor-General, Sir William Slim, has accepted the invitation of the Federal Council of the British Medical Association in Australia to be Patron of the Ninth Session of Congress.

### Plenary Sessions.

The first of the plenary sessions will be held on Tuesday, August 23, from 9.30 a.m. to 12.30 p.m., on the subject of "Cancer". There will be four main speakers: Sir Stanford Cade, Senior Surgeon of Westminster Hospital, London, and a member of the Cancer and Radiotherapy Committee of the Ministry of Health (his subject will be "The Place of Surgery in the Control of Cancer"); Emeritus Professor Sir Peter MacCallum, of Melbourne; Professor E. S. J. King, of the Department of Pathology, University of Melbourne; Dr. C. E. Eddy, of the Commonwealth Radium and X-Ray Laboratory, Melbourne.

Four plenary sessions are listed for the afternoon of Tuesday, August 23.

From 2 to 3.30 p.m. two sessions held simultaneously will be on "The Control of Infectious Diseases" (the speakers to include Dr. H. McLorinan, of Melbourne, Dr. D. W. Johnson, of Brisbane, Dr. C. C. Jungfer, of Lobethal, South Australia, and Dr. J. A. R. Miles, of Adelaide) and on "Rehabilitation" (the speakers to include Dr. G. G. Burniston, of Sydney, Dr. Dudley Longmuir, of Melbourne, and Dr. Selwyn Nelson, of Sydney).

From 3.45 to 5.15 p.m. two sessions held simultaneously will be on "The Use and Abuse of Hormones in General Practice" (the speakers to include Dr. H. P. Taft, of Melbourne, Dr. Adrian Johnson, of Sydney, Dr. J. W. Johnstone, of Melbourne, and Professor Lorimer Dods, of Sydney) and on "Industrial and Occupational Hazards of Health" (the speakers to include Dr. Gordon C. Smith, of Sydney, Dr. Leo J. Gurry, of Melbourne, Dr. J. H. Gowland, of Melbourne, and Dr. Douglas Gordon, of Brisbane).

## DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 2, 1955.[1]

| Disease. | New South Wales. | Victoria.[2] | Queensland. | South Australia. | Western Australia. | Tasmania | Northern Territory.[2] | Australian Capital Territory. | Australia.[3] |
|---|---|---|---|---|---|---|---|---|---|
| Acute Rheumatism | 5(2) | .. | 1 | .. | 1 | .. | .. | .. | 7 |
| Amœbiasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Ancylostomiasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Anthrax | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Bilharziasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Brucellosis | .. | .. | .. | 1(1) | .. | .. | .. | .. | 1 |
| Cholera | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Chorea (St. Vitus) | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Dengue | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Diarrhœa (Infantile) | 14(8) | .. | 3(3) | .. | 2(2) | .. | .. | 1 | 20 |
| Diphtheria | 5(4) | .. | .. | 1 | 1(1) | .. | .. | .. | 7 |
| Dysentery (Bacillary) | .. | .. | 6(4) | 2 | 9(8) | .. | .. | 1 | 18 |
| Encephalitis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Filariasis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Homologous Serum Jaundice | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Hydatid | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Infective Hepatitis | 32(14) | .. | .. | 4(1) | 4(2) | .. | .. | .. | 40 |
| Lead Poisoning | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Leprosy | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Leptospirosis | .. | .. | 1 | .. | .. | .. | .. | .. | 1 |
| Malaria | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Meningococcal Infection | 4(3) | .. | 2(1) | .. | 1 | .. | .. | .. | 7 |
| Ophthalmia | .. | .. | .. | .. | 4(1) | .. | .. | .. | 4 |
| Ornithosis | 1(1) | .. | .. | .. | .. | .. | .. | .. | 1 |
| Paratyphoid | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Plague | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Poliomyelitis | 8(4) | .. | 9(4) | 6(5) | .. | 1 | .. | .. | 24 |
| Puerperal Fever | .. | .. | 1 | .. | .. | .. | .. | .. | 1 |
| Rubella | .. | .. | .. | 2 | 11(6) | .. | .. | .. | 13 |
| Salmonella Infection | .. | .. | .. | .. | 6(6) | .. | .. | .. | 6 |
| Scarlet Fever | 11(4) | .. | 4 | 5(4) | 6(2) | .. | .. | .. | 26 |
| Smallpox | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Tetanus | .. | .. | 2 | .. | .. | .. | .. | .. | 2 |
| Trachoma | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Trichinosis | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Tuberculosis | 47(28) | .. | 19(2) | 7(4) | 8(7) | 4(1) | .. | 1 | 86 |
| Typhoid Fever | 1(1) | .. | .. | .. | 1(1) | .. | .. | .. | 2 |
| Typhus (Flea-, Mite- and Tick-borne) | .. | .. | 3(1) | .. | 1(1) | .. | .. | .. | 4 |
| Typhus (Louse-borne) | .. | .. | .. | .. | .. | .. | .. | .. | .. |
| Yellow Fever | .. | .. | .. | .. | .. | .. | .. | .. | .. |

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Victoria and Northern Territory.

### Membership.

Intending members, especially those desiring accommodation in Sydney, are advised to send in their applications for membership as soon as possible. Application forms are available from the following Honorary Local Secretaries in each State: Dr. M. S. Alexander, 135 Macquarie Street, Sydney; Dr. C. H. Dickson, Medical Society Hall, 426 Albert Street, East Melbourne, C.2; Dr. D. A. Henderson, Ballow Chambers, Wickham Terrace, Brisbane, B.17; Dr. L. Bonnin, 63 Palmer Place, North Adelaide; Dr. S. E. Craig, 7 Malcolm Street, Perth; Dr. K. S. Millingen, 178 Macquarie Street, Hobart.

## Notice.

### THE COLLEGE OF RADIOLOGISTS OF AUSTRALASIA.

THE following candidates were successful in passing the February examination of The College of Radiologists of Australasia, Part II: Dr. P. H. Cody (Victoria), Dr. R. J. Gough (Victoria).

### THE SALK POLIOMYELITIS VACCINE.

As this number of the journal goes to press public announcements are being made on the results of the first extensive use of the Salk poliomyelitis vaccine in the United States of America. It is hoped to include an authoritative comment on the new vaccine in the next issue of the journal.

## Nominations and Elections.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Daly, Lyndsay James, M.B., B.S., 1954 (Univ. Adelaide), 20 Henry Street, Port Pirie, South Australia.

Thompson, John Evelyn, M.B., B.S., 1955 (Univ. Adelaide) (qualified 1954), 16 Roslyn Street, Largs Bay, South Australia.

Sumner, Graham Edwin, M.B., B.S., 1955 (Univ. Adelaide), 92 Portrush Road, Linden Park, South Australia.

Lyall, Allen Raymond, M.B., B.S., 1954 (Univ. Adelaide), 75 Esplanade, Tennyson, South Australia.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Gudkovs, Ariss, qualified 1954; Leske, David, qualified 1954; Mortimer, Isabel Inkster, qualified 1954; Parham, Anthony Robert, M.B., B.S., 1954 (Univ. Adelaide) (qualified 1953); Mussared, Elaine Frances Keele, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Peake, Noel Horace, M.B., B.S., 1954 (Univ. Adelaide); Webster, Victor Henry, M.B., B.S., 1930 (Univ. Melbourne); Westerman, Roderick Alan, M.B., B.S., 1954 (Univ. Adelaide) (qualified 1953).

## Medical Appointments.

Dr. K. V. Sanderson has been appointed honorary clinical assistant, Radiotherapy Department (Skins), Royal Adelaide Hospital.

Dr. Lena Elizabeth McEwan has been appointed surgical registrar in the Royal Adelaide Hospital.

Dr. R. N. Mellor has been appointed ophthalmological registrar in the Royal Adelaide Hospital.

Dr. F. H. Beare has been appointed an honorary consulting physician in the Royal Adelaide Hospital.

Dr. G. A. Lendon has been appointed an honorary consulting physician in the Royal Adelaide Hospital.

Dr. B. F. R. Stafford has been appointed a member of the Nurses and Masseurs Registration Board of Queensland.

The following have been appointed senior medical officers in the Division of Mental Hygiene, Department of Public Health, New South Wales: Dr. Theodora Margaret England, First Medical Officer; Dr. Thelma Black, First Medical Officer; Dr. Mary Patricia Anne Kirton, Medical Officer.

## Deaths.

THE following deaths have been announced:

WISE.—Daniel Wise, on April 1, 1955, at North Essendon, Victoria.

GAULT.—Arthur Kyle Gault, on April 6, 1955, at Adelaide.

ANNETTS.—Henry Allan Annetts, on April 9, 1955, at Sydney.

## Diary for the Month.

APRIL 26.—New South Wales Branch, B.M.A.: Ethics Committee.
APRIL 27.—Victorian Branch, B.M.A.: Branch Council.
APRIL 28.—South Australian Branch, B.M.A.: Scientific Meeting.
APRIL 28.—New South Wales Branch, B.M.A.: Branch Meeting.
APRIL 29.—Queensland Branch, B.M.A.: Council Meeting.
MAY 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

*New South Wales Branch* (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

*Queensland Branch* (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

*South Australian Branch* (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

*Western Australian Branch* (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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